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PEDIATRICS

The Hygienic and Medical Treatment
OF
CHILDREN

BY

CHARLES HUNTER DUNN, M. D.

INSTRUCTOR IN PEDIATRICS, HARVARD UNIVERSITY
PHYSICIAN IN CHIEF AT THE INFANTS' HOSPITAL

FOUNDED UPON THE TEACHINGS OF THOMAS MORGAN ROTCH, M. D.

VOLUME III
Second Edition

THE SOUTHWORTH COMPANY, PUBLISHERS
TROY, NEW YORK
1917

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To

Clarence John Blake, M.D., O.M. (Vienna), F.A.C.S.

Professor of Otology (Emeritus), Harvard University

This Volume is Inscribed

As a tribute to his high professional attainments and to his unfailing
interest in the welfare of children, and in grateful
remembrance of many acts of kindness

By the Author

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DIVISION IX

DISEASES OF THE HEART, PERICARDIUM, AND BLOOD VESSELS

GENERAL CONSIDERATIONS

The diseases of the circulatory system encountered in early life present certain features which differ notably from those of similar pathological conditions in adult life.

ETIOLOGY.—In the adult the pathological conditions affecting the heart and blood vessels may be divided into three groups, as follows: 1. Functional disturbance; 2. infections; 3. degenerative lesions associated with arterial disease. The third group is very important in the pathology of adult life, and includes the conditions primarily due to the wear and tear of life itself, which are represented by arteriosclerosis, and all the degenerative lesions associated with arteriosclerosis, which affect not only the heart, but also the parenchymatous organs throughout the body. This third group is almost non-existent in the pathology of early life. Such pathological conditions as arteriosclerosis, chronic interstitial myocarditis, angina pectoris, aneurism, and "cardio-renal" cases, either are not encountered at all in infancy and childhood, or are so rare as to be pathological curiosities rather than conditions which the physician must consider and be prepared to encounter and treat.

In the place of this degenerative group, there is in early life another group of diseased conditions which assumes great importance in the diagnosis of cardiac disease. This is the group of *congenital cardiac diseases*. It is true that some children with congenital cardiac disease may live until adult life, but many others die in infancy or childhood, and it is in early life that these lesions are chiefly encountered.

The three principal groups, therefore, into which cardiac disease in childhood may be divided are the following:

1. Congenital lesions.
2. Lesions caused by infections.
3. Disturbances of function.

In the second group, there is also a notable difference between childhood and adult life. This is the relatively much greater frequency of *acute infections* involving the heart in childhood. The most common and important cardiac disease of childhood is acute endocarditis; also both acute myocarditis and acute pericarditis are more frequent in childhood than in adult life. The most common cardiac disease of adults, on the other hand, is chronic endocarditis with its associated valvular lesions, and many of the cases trace their origin to an acute infection acquired in childhood.

In the third group, that of functional disturbance, the great difficulty in early life is to draw the line between the physiological and the pathological. The function of the heart is particularly liable to disturbance in early life from comparatively slight causes. These disturbances, however, are associated much less clearly with definite etiologic factors than in adult life, and manifest themselves by symptoms which are subjectively much less marked, and objectively very little removed from the physiological.

SYMPTOMATOLOGY.—Before entering upon a detailed description of the characteristics of cardiac disease in childhood, it is well to consider the particular symptoms of cardiac disease with reference to their significance in early life as compared with adult life.

SUBJECTIVE SYMPTOMS.—Such subjective symptoms as palpitation and precordial pain, while perhaps less common in children than in adults, are nevertheless of more diagnostic significance. In adults, subjective symptoms, while common in organic disease of the heart, are also common in disturbances of a purely functional nature. In children, these subjective symptoms point strongly toward organic disease, the majority of cases of which are of infectious origin. It is true that in some older children of a precocious and neurotic type, subjective symptoms may be a manifestation of functional disturbance only. Usually, however, when a young child *complains* of symptoms referred to the heart, it is strongly suggestive of an organic lesion.

TACHYCARDIA.—In young children the pulse rate is normally rapid, and the rapidity is markedly increased by fever and nervous excitement. Even in older children, particularly in those of a neuropathic type, the frequency of the pulse rate is greatly increased by both exertion and excitement. Under such circumstances the cardiac impulse may become diffuse, and the pulse may become small in volume and easily compressible. The significance of tachycardia as a sign of weakness or dilatation of the heart is consequently very slight in childhood. Occasionally, in older children, when tachycardia and a small compressible pulse are observed only after exertion, it is important to determine whether these signs are only physiological, or are suggestive of cardiac weakness. If after the child has been made to take some moderate exertion, such as walking rapidly up and down the room, or bending the knees deeply ten times while standing, the pulse rate does not return to the normal within three minutes, the possibility of organic weakness of the heart should be considered.

There is a form of paroxysmal tachycardia sometimes seen in older children, which is undoubtedly due to some definite disturbance of function. There are rare cases on record of permanent tachycardia due to pressure on the vagus from enlarged bronchial lymphnodes.

ARHYTHMIA.—This disturbance is much less common in childhood than in adult life as a sign of organic disease of the heart. This is partly because chronic myocarditis is extremely rare in childhood. Also in acute endocarditis, and in cases with the valvular lesions of chronic endocarditis, arhythmia is a rare clinical manifestation in childhood.

Nevertheless, cardiac arhythmia is an extremely frequent finding in early life, usually representing a functional disturbance. It is occasionally physiological in infants and younger children, and can often be noted during sleep in normal children. It is a common manifestation during convalescence in all the acute infectious diseases, particularly after pneumonia and typhoid fever, and under such circumstances is without clinical significance, except in diphtheria. It is a common symptom in severe gastro-intestinal disturbances with toxemia. It is sometimes seen as a symptom of organic disease of the central nervous system, most often in tuberculous meningitis. On the other hand, even in older children, cardiac arhythmia may have no other cause than nervousness; the cause may often be recognized from the fact that the arhythmia disappears after exertion.

BRADYCARDIA.—This symptom is closely associated with arhythmia, and may occasionally be seen as a manifestation of functional disturbance in all the conditions enumerated above, but is much less common. It is also seen in jaundice, and in certain forms of poisoning. Persistent bradycardia, while it may be an idiosyncrasy in some children, is always suspicious of some cerebral disease with increased intracranial pressure.

Bradycardia is rather more common than arhythmia in organic cardiac disease. It is sometimes seen in the toxic myocardial degeneration which accompanies severe acute infectious disease. It is of marked significance after diphtheria. It is sometimes a symptom of the acute infection of the myocardium seen in rheumatic fever. Attacks of typical heart block are associated chiefly with the degenerative lesions of adults, and are very rare in early life.

DYSPNEA.—This symptom has much the same significance in children as in adults. If the various causes of dyspnea outside the heart can be excluded, dyspnea usually means organic cardiac disease, either infectious or congenital. It is a notable fact that dyspnea is less common as a manifestation of acute endocarditis in adults than in children. In the former, acute endocarditis is apt to develop insidiously, without dyspnea or subjective symptoms, while in the latter, acute endocarditis is apt to be accompanied by distinct symptoms of cardiac weakness.

CYANOSIS.—Persistent cyanosis in early life usually means congenital cardiac disease, although it is sometimes seen in very young infants as a sign of the congenital pulmonary atelectasis described under diseases of the newborn. Cyanosis is uncommon in early life as a sign of the cardiac diseases due to infection. It is sometimes seen in certain diseases of the lungs, but is less common in such conditions than in adults.

EDEMA AND OTHER SIGNS OF CHRONIC PASSIVE CONGESTION.—Cardiac edema in childhood is mainly seen in cases of chronic valvular disease. In such cases it has the same clinical significance in early life as in adult life. As uncompensated chronic valvular disease of the heart is less common in children than in adults, and as the degenerative lesions causing cardiac weakness and edema are almost unknown in children, edema is a relatively less common clinical manifestation in early life. It is an uncommon symptom of the acute infections of the heart so frequently seen in childhood, even though the lesions produce other notable signs of cardiac weakness.

Enlargement and tenderness of the liver is an important sign of cardiac insufficiency, and is seen in chronic valvular disease under the same circumstances as edema. It is more common than edema as a sign of cardiac weakness in acute endocarditis.

Cough is one of the most important symptoms of chronic passive congestion from cardiac insufficiency seen in early life. It is an almost constant symptom of this condition, whether produced by acute infection or by chronic valvular disease.

HEART SOUNDS AND MURMURS.—In infancy the first heart sound is normally short and dull, and only at a later age is heard the loud first sound which is characteristic of childhood.

Murmurs are a very common clinical finding in childhood. In the first two or three years of life, however, heart murmurs are comparatively uncommon, and when present are usually a sign of congenital organic disease of the heart.

The cardiac murmurs heard in early life may be divided into four etiologic groups, as follows: 1. Accidental murmurs; 2. murmurs due to functional insufficiency; 3. murmurs due to congenital cardiac lesions; 4. murmurs due to acquired valvular lesions. This classification is designed to avoid the confusing terms "organic" and "functional" as applied to cardiac murmurs. It is true that in the last two groups the murmurs are due to true organic lesions. In the second group, the essential condition is a functional insufficiency which may or may not be caused by a demonstrable lesion, which leads to a dilatation and relative insufficiency. In the first group there is no evidence that the murmurs are due either to organic lesions or to a disturbance of the function of the heart.

ACCIDENTAL MURMURS.—These murmurs are extremely common in childhood and constitute the majority of the cardiac murmurs heard in early life. They are practically unknown in infancy, and are rare in the third and fourth years. They are commonest during the school age, the same age at which the murmurs due to valvular disease are found. In school children, accidental murmurs greatly preponderate over organic murmurs. Some authors declare that they can be found at some time in one-half or even in three-quarters of all school children.

These accidental murmurs are systolic in time, and are usually of a soft blowing character. They are heard all over the precordia, and are loudest either at the left border of the sternum near the nipple, or at the base. They never replace the first heart sound, and usually begin at about the middle of systole. They are not transmitted into the axilla or into the vessels of the neck. They are usually inconstant in occurrence. They are very apt to change in character, or to disappear entirely when the position of the child is changed. Often they are increased in intensity when the child is placed on its back, and sometimes may be heard only when it is in that position. They are often intensified by increased action of the heart, whether due to fever, exertion, or excitement. They are also often increased by deep inspiration, and diminished during expiration, especially if expiration be forcible. They sometimes disappear entirely when the child holds its breath, particularly if the breath be held at the end of expiration. The most important point of all in the recognition of accidental murmurs, is that *they are unaccompanied by any increase in the area of cardiac dulness, or by any symptoms referable to the heart and circulation.*

The cause of these accidental murmurs is not yet definitely established. The majority of writers regard most of them as cardio-pulmonary in origin. Under this explanation the lingula of the left lung which is in close juxtaposition with the heart is affected by the movement of cardiac systole. The explanation of the production of the murmur by this movement involves various theories, such as aspiration of air, or a friction rub, or that the sound is produced by the movement of the lung tissue. In favor of the extra-cardiac origin of accidental murmurs is their inconstant character, and their modification by cardiac overexertion, by changes in the respiration, and by changes of position. While it is probably true that a large number of these murmurs are cardio-pulmonary, it is also probable that this explanation does not hold for all the murmurs designated as accidental.

It is probable that some of the murmurs which present the clinical features of the accidental group, belong etiologically in the group of murmurs due to functional insufficiency. In these cases, if a dilatation of the heart leading to mitral insufficiency is present, it is too slight to be detected on physical examination or to produce symptoms of cardiac weakness. It is possible that there may be no dilatation, but a relative insufficiency of the papillary muscles attached to the mitral valve. Whatever the underlying condition, the immediate cause of the murmur is the same as in the group due to relative cardiac insufficiency, namely, a relative incompetence of the mitral or tricuspid valves. The only difference is clinical. Murmurs of this class have been designated as "atonic." Many of the murmurs heard in the course of acute febrile diseases belong in this category. They are also common in delicate nervous children.

Another suggested cause for the accidental murmurs is an increased fluidity of the blood, with a consequent increased velocity of flow. This is the explanation of the so-called anemic murmurs. Anemia is probably the commonest cause of the accidental murmurs heard in adults, but in childhood I believe this cause to be comparatively rare. Because a heart murmur is heard in an anemic child, it is no proof that the murmur is due to the anemia. There are cases on record of children with grave anemia in whom the murmur disappeared after transfusion. On the other hand, a heart murmur is by no means a constant symptom of anemia in early life, and many children suffering from grave anemias have no murmurs. I believe that the rôle of anemia in the accidental murmurs of childhood has probably been exaggerated.

Another theory brought forward to explain the accidental murmurs, is that of a relative widening of the pulmonary artery in childhood, which, in connection with the narrower ventricular opening, causes a murmur. There is no definite evidence in support of this theory.

The diagnosis of accidental cardiac murmurs in childhood is of great importance. Their character is such that there is no difficulty in distinguishing them from the murmurs due to congenital lesions. They are distinguished from the murmurs due to functional insufficiency by the absence of the symptoms and physical signs of cardiac weakness and dilatation, although, as stated above, some of them belong etiologically in the functional insufficiency class. The chief difficulty is in distinguishing them from the murmurs due to compensated valvular disease. When the physical examination shows signs of compensating cardiac hypertrophy, the recognition of an organic endocardial murmur is not difficult. Unfortunately, in mitral insufficiency from chronic endocarditis a considerable time often elapses before the characteristic enlargement of the area of cardiac dulness and accentuation of the pulmonic second sound become evident. In such cases the differentiation of accidental from endocardial murmurs becomes extremely difficult, and prolonged observation with repeated examinations may be necessary before a diagnosis can be made. If the murmur possesses any of the characteristics mentioned above as typical of accidental murmurs, such as inconstancy, change with respiration or position, failure of transmission, and so forth, the probabilities are strongly in favor of its accidental nature. If on the other hand there is a previous history of possible rheumatic fever infection, or if the tachycardia produced by moderate exertion does not quickly subside, there is a probability of valvular disease.

In my experience many more cases of accidental murmur are diagnosed as cases of endocarditis than vice versa. This is true mainly because the very great frequency of accidental murmurs in childhood is forgotten. This mistake leads to a very unnecessary and inadvisable restriction of the normal activities of the growing child. Many physicians recommend such restriction in doubtful cases in order to be "on the safe side." While I believe in taking every necessary precaution against cardiac overstrain, I further believe, as I shall explain under Endocarditis, that the tendency has been to exaggerate restriction of activity in childhood even in chronic valvular disease. When a cardiac murmur is of doubtful nature, if the normal activities of the child's life produce absolutely no symptoms or signs of cardiac insufficiency, I do not believe that the mere presence of a murmur is an indication for restricting those activities. In many such cases the murmur will eventually prove to be accidental, and even if the final diagnosis should be valvular disease, no harm will come from permitting normal activity provided that all extreme overexertion be avoided, and that the child be kept under close observation.

MURMURS DUE TO FUNCTIONAL INSUFFICIENCY OF THE HEART.—The cause of these murmurs is a disturbance of the balance between the functional power of the heart, and the work which the heart is required to perform. Such a disturbance of balance may be produced on the one hand by causes acting on the heart in such a way as to diminish its muscular strength, and on the other hand, by an abnormal increase in the load thrown upon the organ. Obviously the functional power of the heart can be diminished by the existence of valvular disease, but in such a case the murmur is due to the valvular lesion, and belongs in the group described below. An acute infection of the myocardium also produces a functional insufficiency, but acute myocarditis is rare without an accompanying endocarditis, and consequently the murmurs heard in acute infections of the heart are usually produced by the lesions of the valves. The heart muscle may, however, be affected by the toxins produced by acute infections in other parts of the body. The process in such a case is in the nature rather of an acute degeneration of the heart muscle than of an inflammation. The commonest diseases in which this toxic degeneration occurs are diphtheria and scarlet fever. The result of the process is weakening of the heart muscle, impairment of its efficiency, and dilatation of the ventricles. The result of the dilatation is a relative insufficiency of the mitral or tricuspid valves, which manifests itself by a systolic murmur. In these cases the murmur is not a manifestation of primary cardiac disease, but only of a secondary cardiac insufficiency produced by disease elsewhere.

In some children, the development of the heart does not keep pace with the growth of the child. This is particularly common in children who grow very rapidly. The result is that the heart becomes relatively insufficient to cope with the load which is thrown upon it; slight dilatation may occur, and a relative mitral or tricuspid insufficiency may cause a leak sufficient to produce a murmur. The same condition may be produced by violent overexertion, even though the heart be sufficient to deal with a normal load. This is a primary cardiac condition and will be described in further detail under the functional disturbances.

The chief characteristic of these murmurs is their temporary character. Whether the cause be myocardial degeneration in acute infections, or relative overload, the murmur disappears with the cause. In the acute infections these murmurs disappear in convalescence, persisting longest in diphtheria. In the rapidly growing child they disappear with reduction of the load, or in the course of time.

These murmurs are systolic in character. They are sometimes difficult to distinguish from the murmurs of valvular disease. The chief point is their soft character, and the usual absence of transmission into the axilla. The diagnosis eventually rests upon their disappearance. They are still more difficult to distinguish from the accidental murmurs. The only feature by which they can be distinguished from the accidental murmurs is the presence of an increased area of cardiac dulness, and the frequent coincident presence of other signs of cardiac weakness.

MURMURS DUE TO CONGENITAL LESIONS.—In this group belong most of the murmurs heard in the first two or three years of life. Acquired cardiac disease and functional insufficiency are rare at this age, and accidental murmurs are almost never heard. Murmurs caused by congenital disease may also be present in the later years of childhood, and it is often necessary in diagnosis to distinguish them from the murmurs of acquired valvular disease. In the differential diagnosis, the following features are in favor of congenital disease: A very loud, harsh systolic murmur with a normal area of cardiac dulness; any murmur in the first three years with an increased area of cardiac dulness; a loud harsh murmur heard only at the base of the heart; a "humming-top" murmur, that is, a murmur heard throughout the cardiac cycle; association with the murmur of cyanosis, or a palpable systolic thrill.

MURMURS DUE TO ACQUIRED VALVULAR LESIONS.—These are the murmurs characteristic of acute and chronic endocarditis. They are uncommon before the fourth year of life. They have the same characteristics as the murmurs due to valvular disease in adults. The commonest is the systolic murmur of mitral insufficiency, and second in frequency is the presystolic murmur of mitral stenosis. Aortic disease, while less common than mitral disease, does occur as a manifestation of endocarditis in childhood, but the murmurs of aortic insufficiency and stenosis are much less common in children than in adults.

The nature of these murmurs is usually recognized with comparative ease. The systolic murmur of mitral insufficiency must be distinguished from congenital murmurs, accidental murmurs, and murmurs due to functional insufficiency. The differentiation from congenital murmurs has already been described. The important feature in the recognition of systolic murmurs due to acquired valvular disease is the association of the murmur either with signs of cardiac dilatation or hypertrophy, or with fever and the symptoms of cardiac weakness, when causes other than rheumatic fever can be excluded. The transmission of the murmur into the axilla is also a characteristic of murmurs due to valvular disease in children as it is in adults.

THE PULMONIC SECOND SOUND.—Accentuation of the pulmonic second sound is often found in healthy children, and is not of *so much* diagnostic significance in childhood as in adult life. It can be caused by exertion, excitement, or crying. Its presence does not mean very much in diagnosis; but its absence is strongly against the diagnosis of chronic endocarditis.

THE VENOUS HUM.—This sign is fairly common in older children. It is sometimes heard in the vessels of the neck, but in seeking it care must be taken to avoid pressure with the stethoscope. A particularly common site in children is both sides of the upper part of the sternum. In this situation is often heard a low, long-drawn, continuous hum, which may be quite loud, which is increased during systole, and which can only be explained by an origin in the great veins. The sound often becomes weaker when the patient lies down. It is frequently particularly loud to the right of the sternum at the aortic auscultation area.

The cause of this sound is not certainly known. It is found most often in anemic children, and its explanation in such cases is increased fluidity and rapidity of flow of the blood. It is sometimes found in children who are not anemic, and in such cases the cause is obscure. Pressure on the veins from enlarged mediastinal lymphnodes has been suggested as a possible cause in some cases.

DIAGNOSIS.—The general diagnosis of the cardiac diseases present problems which are as a whole probably less difficult in childhood than in adult life. This is mainly due to the fact that many of the conditions most difficult of diagnosis in adults do not occur in early life. The unimportance in childhood of the great group of chronic degenerative lesions associated with arterial disease has already been mentioned. Furthermore, the functional disturbances of the heart in childhood are less numerous, have a less multiform etiology, and are easier of recognition. The absence of alcohol and tobacco as etiologic factors is an example of the difference.

The diagnosis of the cardiac diseases in early life must depend mainly on old and well established clinical methods. It is to the results of a careful history and physical examination that we must chiefly look in making a diagnosis. Laboratory methods of diagnosis have comparatively little place in this division of the domain of pediatrics.

The same is true of the recent great advances made in the study of the circulatory diseases of adult life, through the application of physiological methods to clinical investigations. The most important new device for the study of the diseases of the heart is the electrocardiograph. This instrument has led to an entirely new classification of a certain group of cardiac disturbances in adults. Whether or not the electrocardiograph will throw any very important light upon the diseases of the heart in childhood is yet to be determined. Children have not yet been extensively studied with this instrument, probably because the disturbances of adults presented a much more promising field. Most of the disturbances upon the nature of which light has been thrown by investigation with the electrocardiograph, do not occur in early life. Furthermore this instrument has not been, and probably cannot be, adapted to general clinical use, and investigations with it can only be carried out in institutions.

The various instruments for the estimation of the blood pressure in the arteries are also of comparatively little value in the diagnosis of the cardiac diseases of early life. Blood pressure investigations have been extensively carried out in children, but the results have been disappointing from the point of view not only of diagnosis, but of indicating treatment. The normal standards are widely variable, and the changes seen in the various diseased conditions are inconstant. The verdict appears to be that testing the blood pressure in children is not an important or essential diagnostic procedure.

The most important of the special methods of investigation applicable to the diagnosis of cardiac disease in early life is examination with the roentgen ray.

The prognosis and treatment of the diseases of the heart, pericardium and blood vessels in early life, vary so much in the different pathological conditions, that no generalizations can be made.

I. CONGENITAL CARDIAC DISEASE

Congenital cardiac disease is a condition of anomaly in the anatomical structure of the heart, having its origin in intrauterine life.

ETIOLOGY.—The cardiac anomalies may be divided into two chief groups. Those due to *arrest of development*, and those due to *fetal endocarditis.*

ARREST OF DEVELOPMENT.—This may occur at an early stage, before the different parts of the heart have been fully formed, or it may occur immediately after birth. In the former case, the anomaly is present in the fully developed fetal heart. In the latter case the fetal heart has been fully developed at the time of birth, the arrest of development consisting of a failure of the changes which normally take place in the anatomy of the fetal circulation immediately after birth, leading to a persistence of some condition which is normal in the fetus.

The underlying causes of arrested development of the heart have always been obscure, and at the present day there is little definite knowledge. The most important fact bearing on the ultimate etiology of imperfect development of the heart is the frequency of association of congenital cardiac lesions with developmental anomalies involving other parts of the body. While the figures of different reported series of cases differ widely, the frequency of association of congenital cardiac lesions with grave malformations in other parts of the body is too great to be considered accidental.

The weight of modern opinion inclines to the view that the cause of all developmental malformations is to be sought, not in a hereditary predisposition, but in a diseased condition of the fetal envelopes or maternal tissues. This view is based on the evidence supplied by the family history of these cases. A history of any congenital disease in the ancestry is much less common than is one of congenital disease in other members of the same generation. The most common evidence in the history having a possible bearing on congenital malformations is that of injurious influences acting upon the mother, or within her organism. Evidence of infection in the mother is present at times, and it is probable that maternal syphilis is one of the most frequent causes. Other possible causes often found in the family histories of cases of malformation, are various influences acting upon the mother in the earlier weeks of pregnancy, such as fright, worry, or ill-treatment, or a condition of particularly unfavorable hygienic surroundings.

While such statistical evidence is not very conclusive, it is all that we have in explanation of congenital malformations. I believe that the chief cause must be sought in conditions which affect the immediate environment of the developing embryo. Heredity, however, cannot be wholly excluded as an etiological factor in some cases.

For those cases in which the arrest of development occurs immediately after birth, the lesion consisting of a persistence of a condition normal in the fetal circulation, a somewhat different explanation must be sought. In the majority of cases these lesions are associated with other cardiac lesions dating from an earlier period. These earlier lesions produce a disturbance in the circulation, which becomes marked under the conditions attending the change from the fetal circulation to that of the newborn infant. An increased pressure may be produced in some of the chambers of the heart. The result of these circulatory disturbances is a persistence of certain fetal conditions, which persistence really represents an effort to offset or compensate for the effect produced by the associated lesions. It is obvious, for example, that a stenosis or atresia of the pulmonary artery would not have a deleterious action during fetal life, but that upon birth, when the lungs are expanded, and the pulmonary circulation comes into play, the result of such a lesion would be a marked circulatory disturbance, with backing up of blood in the right cardiac chambers. If now either or both of the two normal fetal outlets from the right side of the heart, namely, the *foramen ovale* and the *ductus arteriosus Botalli*, persist, the effect will be compensatory, and the circulatory disturbance produced by the pulmonary stenosis is certainly the probable cause of their persistence.

This does not explain the occasional cases in which the persistence of a fetal condition is the sole cardiac lesion. The reports on which our knowledge of congenital cardiac disease is properly based, include mainly records of cases in which post-mortem examinations were made. I have seen a number of cases in which the clinical signs of patent ductus arteriosus were clear in early infancy, but disappeared in later infancy or childhood. I believe that temporary persistence of a fetal condition as the sole lesion is commoner than would be supposed from post-mortem statistics and that it represents only a delay in the process by means of which the fetal openings are normally closed. The cause of the delay seems obscure at first sight, but irregularities in the time of development of various anatomical structures throughout the body are not uncommon, and there is no reason why they should not sometimes involve the heart of the newborn infant.

In the rare cases in which a fetal condition persists throughout life as the sole cardiac lesion, the etiology is wholly obscure.

FETAL ENDOCARDITIS.—There are a certain number of cases in which the anatomical evidences of endocardial thickening and cicatricial contraction demonstrate that the lesion was produced by an inflammatory process in the fully developed fetal heart. Such cases are undoubtedly due to fetal endocarditis. Formerly acute endocarditis was considered a very important etiologic factor in congenital cardiac malformation, and not only the cases showing clear evidences of its action on the fully developed heart, but also cases in which the character of the lesion suggested arrest of growth, were explained on this basis. In particular, stenosis and atresia of the pulmonary artery were considered inflammatory in origin. This view held that these and similar lesions were produced by the action of acute infection upon the partly developed heart.

The weight of modern opinion, with increasing knowledge of development and its phenomena, is inclining toward the view that in most of the cases showing such lesions as pulmonary stenosis, the essential cause is arrest of growth, rather than cicatricial contraction. The imperfect development is produced by the causes enumerated above under arrested development. The presence of thickening of the endocardium without cicatricial contraction is no proof of a previous inflammatory process. It seems better to consider that fetal endocarditis is the cause of the lesion only in those cases in which the evidences of an inflammatory process in the fully developed fetal heart are clear.

PATHOLOGICAL ANATOMY.—From what has been said above in connection with the etiology of congenital cardiac disease, it is evident that the physician, in order to understand the various lesions, must be familiar, not only with normal development in the newborn infant, but must also have some knowledge of the normal development of the fetus. The three stages of normal development having a special bearing on the congenital cardiac lesions are the following: 1. The development of the fetal heart; 2. the fully developed fetal circulation; 3. the changes in the circulation occurring at birth.

DEVELOPMENT OF THE FETAL HEART.—If it were necessary that the pediatrist should be familiar with every possible form of congenital cardiac anomaly, he would have to be thoroughly familiar with all the facts known about the embryonic development of the heart. Although a very great variety of cardiac lesions have been reported, most of them are so extremely rare as to be pathological curiosities rather than lesions of clinical importance. For this reason only the few facts concerning the development of the heart which are necessary in explaining the commoner congenital lesions, will be enumerated here.

The heart is originally formed by two straight tubes, which merge together, with complete disappearance of the septum between, forming a single chamber. The stages of development most intimately connected with the commoner congenital anomalies are those concerned with the formation of the septa which come to separate the several chambers of the heart, and with the formation and division of the *truncus arteriosus*, from which the primitive aorta and pulmonary artery are derived.

The division of the auricles occurs through the development of two different septa, placed in planes parallel with each other, and called respectively the *septum primum* and the *septum secundum.* At one stage of development the septum primum has two openings, one, the *ostium primum,* at its lower border, the other, the *ostium secundum,* in the upper posterior portion. The ostium primum closes at the end of the fifth week. The septum secundum, developing somewhat later, arises from the upper wall of the auricle, in a plane a little to the right of the septum primum. Passing downward it covers in the upper and anterior portion of the ostium secundum, giving it a valvular character, and transforming it into the *foramen ovale.*

The *truncus arteriosus* is formed partly from the primitive aortic arches, and partly by the distal portion of the *bulbus cordis.* The latter is a transitory portion of the embryonic heart leading from the right end of the common ventricle to the aortic arches. Its proximal portion becomes the termination of the ventricle, while its distal portion becomes part of the primitive aortic trunk. The division of the truncus arteriosus into aorta and pulmonary artery occurs through the development of the aortopulmonary septum, derived from two septa. One begins to develop at the junction of the fourth and sixth aortic arches, and grows downward, dividing the lumen of the truncus transversely, the aortic lumen being the one in front. The other is derived from certain endocardial swellings in the bulbus cordis, which are arranged spirally, and form a spiral bulbar septum. The spiral arrangement has the effect of torsion of the great vessels, bringing the lumen of the aorta behind and to the left, and that of the pulmonary artery in front and to the right. The aortopulmonary septum is completed by the junction of these two septa, and the division is further completed by junction of the aortopulmonary septum with the interventricular septum.

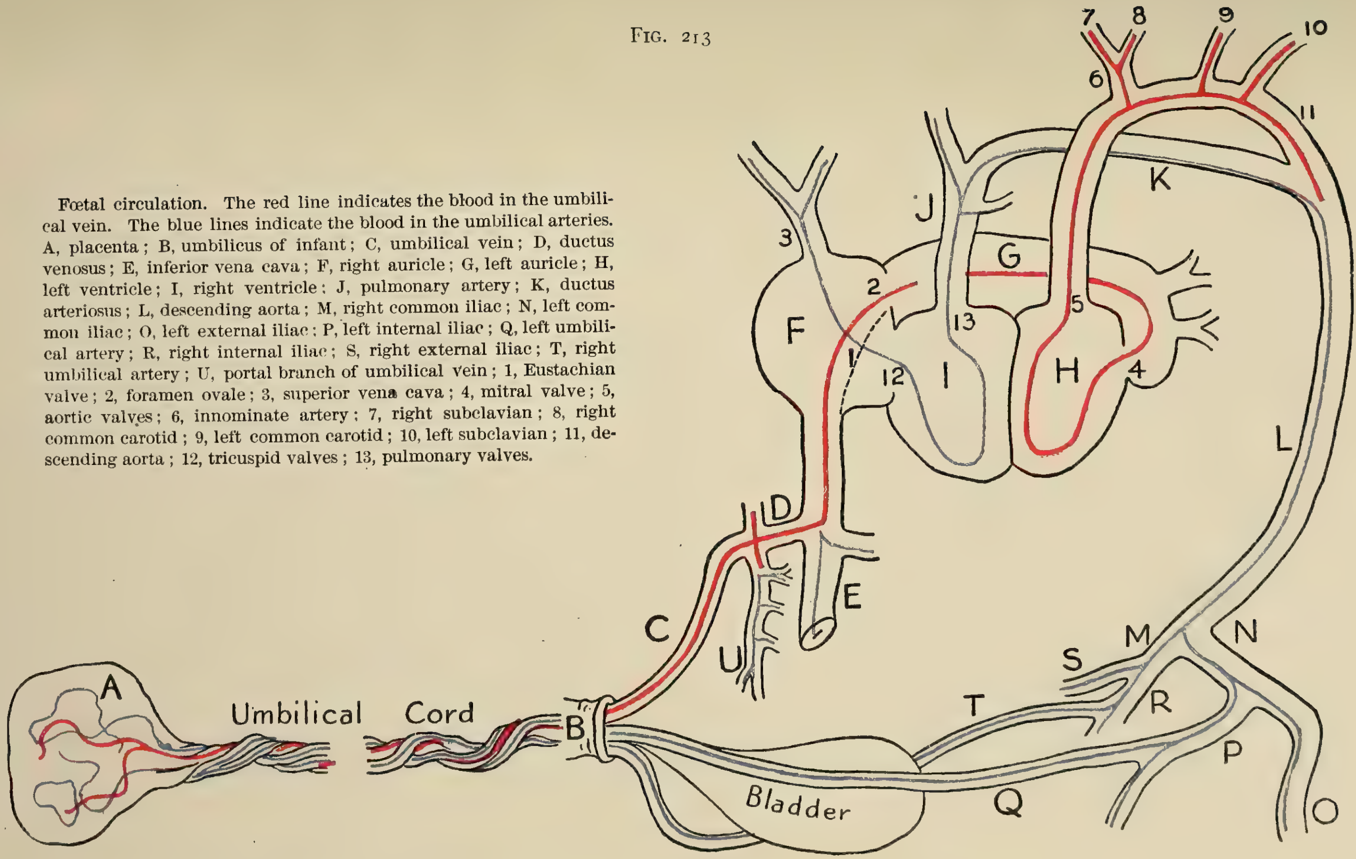
The *intraventricular septum* begins to develop at about the fourth week as a crescentic ridge on the lower wall of the ventricle. It grows upward and backward, and unites with the prolongation of the proximal aortic septum, thus completing the division of the heart into right and left, the arterial and venous openings being each situated in its proper ventricle.

These are the essential facts, stripped of details, which are important in explaining the commoner congenital anomalies.

THE FETAL CIRCULATION.—The chief anatomical change which takes place at birth is the transition from the intrauterine circulatory mechanism to a form adapted to extrauterine life; in other words, from the oxygenation of the blood through the placenta to the same process carried on by the lungs. A general knowledge of the fetal circulation is, then, evidently of considerable importance, especially when it is considered that a large proportion of the cases of congenital heart disease which we are called upon to diagnose is represented by defects which prenatally were perfectly normal conditions, such as an open ductus arteriosus, or a patent foramen ovale.

Fig. 213 represents the course of the (red) oxygenated blood from the placenta to the infant, and that of the darker (blue) deoxidized blood from the infant back to the placenta. We must consider that in the fetus the lungs are in a collapsed, inert condition, performing no part in the fetal economy, but remaining quiescent until called upon to perform their special function at birth. The true lung of the fetus, therefore, is represented by the placenta of the mother. It is here that the blood is oxygenated, and is carried by means of the umbilical vein directly through the umbilicus of the fetus to the liver, as is seen in the diagram. In the liver, the umbilical vein divides into three branches: 1. The smallest, carries the blood directly to the liver tissue, whence it is returned as in the adult to the inferior cava by the hepatic veins; 2. the largest portion meets and mixes with the blood from the portal system, and is distributed with it to the liver; 3. the remaining portion is carried, by a vessel called the *ductus venosus*, directly to the inferior cava, where it meets the deoxidized blood from the lower extremities, mixes with it, and is carried to the right auricle. Here, instead of passing as in the adult into the right ventricle, it is directed by the *Eustachian valve* through the *foramen ovale*, into the left auricle. It then passes into the left ventricle through the mitral orifice, and thence through the aortic orifice into the aorta. The greater part of this blood-current is then carried by the carotid and subclavian arteries to the head and upper extremities, where, after doing its work in vitalizing the tissues and taking up their waste (a small portion also passing, as usual, into the descending aorta), it is returned as deoxidized blood through the veins to the superior cava into the right auricle, thence through the tricuspid orifice into the right ventricle, and up through the pulmonary artery, where a small portion is distributed as usual to the lungs, while the main portion is carried directly over to the descending aorta by the *ductus arteriosus Botalli*. It here mixes with the small portion of oxygenated aortic blood mentioned above, and passes down the aorta, being distributed on its way, as in the adult, until it reaches the internal iliac arteries. From these arteries it is carried, by branches called the *umbilical arteries*, through the umbilicus to the cord and placenta.

FIG. 213



Fœtal circulation. The red line indicates the blood in the umbilical vein. The blue lines indicate the blood in the umbilical arteries. A, placenta; B, umbilicus of infant; C, umbilical vein; D, ductus venosus; E, inferior vena cava; F, right auricle; G, left auricle; H, left ventricle; I, right ventricle; J, pulmonary artery; K, ductus arteriosus; L, descending aorta; M, right common iliac; N, left common iliac; O, left external iliac; P, left internal iliac; Q, left umbilical artery; R, right internal iliac; S, right external iliac; T, right umbilical artery; U, portal branch of umbilical vein; 1, Eustachian valve; 2, foramen ovale; 3, superior vena cava; 4, mitral valve; 5, aortic valves; 6, innominate artery; 7, right subclavian; 8, right common carotid; 9, left common carotid; 10, left subclavian; 11, descending aorta; 12, tricuspid valves; 13, pulmonary valves.

CHANGES IN THE CIRCULATION AT BIRTH.—At birth the expansion of the lungs opens a new path for the circulation through the pulmonary arteries, and the blood ceases to pass through the foramen ovale and ductus arteriosus. With the ligation of the cord, circulation through the umbilical vein and ductus venosus ceases, and these vessels become filled with thrombi, which later undergo fibrous tissue organization. By the end of the first week these vessels are closed, at their extremities at least, and finally they become small fibrous cords. The ductus arteriosus also becomes the seat of a proliferating endarteritis, which, with organization of the clot, converts the vessel into a fibrous cord. It is rarely open after the tenth day. The closure of the foramen ovale takes place through the development of a membrane, which requires more time, so that complete closure may not occur until the middle of the first year.

LESIONS.—A very large number of congenital lesions of the heart have been reported in medical literature. For a complete list, the reader is referred to Maude E. Abbott's article on Congenital Diseases of the Heart, in Vol. IV. of Osler and McCrae's Modern Medicine. In this list about 64 different congenital anomalies are classified, based on an analysis of 631 cases with the clinical symptoms and post-mortem findings. The majority of these lesions are so rare as to be pathological curiosities, and their clinical manifestations do not afford a sufficient basis for definite diagnosis. There are, however, a certain number of congenital cardiac lesions which are comparatively common, and which constitute diseased conditions which the physician should be prepared to recognize, diagnose, and treat.

Among these lesions *patent foramen ovale* is one of the most common. It represents persistence of a condition which is normal in the fetus, and is usually compensating, that is, the opening is kept in existence because of the presence of some other lesion which so embarrasses the circulation that the extra opening in the interauricular septum is necessary. Occasionally an open foramen ovale is found as the sole lesion. In such cases I believe the cause is truly developmental.

Small oblique openings in the foramen are not uncommon in autopsies upon young infants, but have no pathological significance. In pathological cases the opening is much larger, measuring from one-quarter to one inch in diameter.

Although an open foramen ovale is a comparatively common lesion, it is very unimportant from the clinical point of view. When associated with other lesions, it does not betray itself by any special symptoms, and its additional presence has no bearing on prognosis. When the sole lesion it may or may not betray itself by the presence of a murmur, and there are no other symptoms. Consequently, an open foramen ovale can rarely be diagnosed, does not present a disease picture, and will not be considered as a distinct disease.

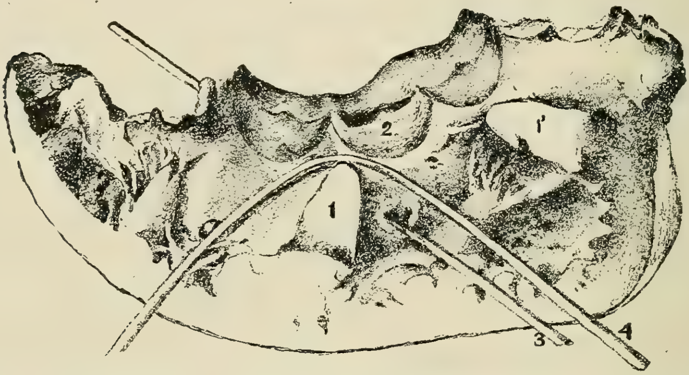
The common congenital lesions which are of clinical importance are the following:

1. Stenosis or Atresia of the Pulmonary Artery.
2. Defect in the Interventricular Septum.
3. Persistence of the Ductus Arteriosus Botalli.
4. Transposition of the Great Vessels.

STENOSIS OF THE PULMONARY ARTERY

This is generally stated to be the most common congenital anomaly of the heart, and on the basis of post-mortem findings, the statement is true. From the clinical point of view, pulmonary stenosis is the most important congenital cardiac lesion.

FIG. 214



Congenital cardiac disease. Male 4½ years old. Right and left ventricles laid open by two cuts. Stenosis of pulmonary orifice. Incomplete septum ventriculorum. 1 and 1', septum ventriculorum cut across; 2, aortic valves; 3, probe passing through narrowed pulmonary orifice; 4, bent probe, passing through right ventricle to left through opening in septum ventriculorum.

PATHOLOGICAL ANATOMY.—Two types of lesion are found. In the first the stenosis is always valvular in character, and is produced by thickening, shortening, or fusion of the pulmonary cusps. In this type the pulmonary stenosis is usually the only congenital anomaly which is found. In the second type, which is much more common, while there may or may not be thickening of the valve cusps, the essential lesion is a rudimentary condition or hypoplasia of some part of the pulmonary tract. The hypoplasia may involve only the valvular orifice, although more often the conus shares in the narrowing. There may be a point of narrowing above the valves. There may be a hypoplasia of the whole pulmonary tract, including the artery. In any part of the tract the narrowing may amount to a complete atresia. With this second type of lesion are usually found other congenital cardiac anomalies, such as a defect in the interventricular septum, an open ductus arteriosus, or an open foramen ovale.

ETIOLOGY.—The first type probably represents the results of a fetal endocarditis acquired after the heart was fully formed. The second type undoubtedly represents a developmental anomaly acquired early in fetal life.

SYMPTOMS.—The principal symptoms are *cyanosis*, a *systolic murmur*, a *systolic thrill*, and *enlargement of the area of cardiac dulness* to the right.

Cyanosis is the most important and constant symptom of all forms of pulmonary stenosis. It is so constantly present in this disease that without it a diagnosis cannot be made. Not only is cyanosis almost constant in pulmonary stenosis, but it is a rare symptom in other forms of congenital cardiac anomaly, so that its presence is always strong evidence of an obstruction to the flow of blood through the lungs. The principal other congenital lesion in which cyanosis is found is a rare one, namely, complete transposition of the great vessels. Apparently the mere admixture of venous with arterial blood through openings in the cardiac septa, or through an open ductus arteriosus, is not sufficient to produce cyanosis, provided that there is no interference with the flow of blood through the lungs. It is with such an interference that cyanosis is particularly associated.

The cyanosis is present from birth. It varies in degree. The bluish discoloration of the skin and mucous membranes may only be notable or marked when the infant cries, or it may be constantly present. In cases of stenosis without complete atresia, it is most marked when the interventricular septum is open. In cases with complete atresia it is still more intense, especially when the septa are closed. Polycythemia is always present in permanent congenital cyanosis, the red count usually lying between 7,000,000 and 10,000,000.

The murmur is systolic in time, is harsh in character, and is usually heard loudest at the second left interspace, and along the left border of the sternum. The pulmonic second sound, except in cases with an open ductus arteriosus, is weak or absent. A murmur is absent in two conditions when there is no associated defect in the interventricular septum and no open ductus arteriosus: First, when there is complete atresia of the pulmonary artery; second, when there is general hypoplasia of the pulmonary tract without narrowing at any one point.

The systolic thrill is not palpable in all cases. Its absence does not exclude pulmonary stenosis, but its presence in association with cyanosis is diagnostic.

The enlargement of the area of cardiac dulness is due to compensatory hypertrophy of the right ventricle. It is usually not present at birth, but develops during the early weeks in infants who survive. Theoretically the enlargement should be chiefly to the right. In early life, when there is hypertrophy of the right ventricle, the heart tends to swing a little toward the left, so that in infants and children enlargement of the area of cardiac dulness is usually found to the left as well as to the right, with displacement of the impulse to the left. This point must be remembered in interpreting percussion findings in early life. Evidences of cardiac enlargement are practically a constant finding in long standing cases of pulmonary stenosis.

The other principal symptoms of pulmonary stenosis are dyspnea, suffocative attacks, attacks of marked weakness or faintness, and a tendency toward infection of the lungs. These symptoms vary in their presence or absence, and in their severity, in different cases. The persistent cyanosis always eventually produces *clubbing of the finger tips.*

COURSE.—The course of the disease varies according to whether or not complete atresia is present, and according to whether or not either a defect in the septa (open foramen ovale or open interventricular septum), or a patent ductus arteriosus, is present as an associated lesion. In complete atresia death usually occurs very shortly after birth. A few cases have survived longer, the lesion being partly compensated by an opening in the septum or by an open ductus arteriosus.

When pulmonary stenosis is the sole lesion, a very real mechanical obstacle is introduced into the circulation through the backing up of the blood in the right side of the heart without any other outlet. This interference with the circulation is usually incompatible with life, and most of these cases die very shortly after birth. When the interventricular septum, the foramen ovale, or the ductus arteriosus remains open, these lesions are secondary to the pulmonary stenosis, and an outlet is provided from the right side of the heart into the aortic circulation. This at least partly relieves the mechanical interference with the circulation, and acts as a compensatory condition. A few cases of pulmonary stenosis are on record which survived in spite of the absence of any of these secondary compensatory lesions. Usually, however, when a case showing the clinical evidences of pulmonary stenosis survives the early weeks of life, one or more of these secondary lesions are present. Survival is consequently an important point in the diagnosis of the association of other lesions with pulmonary stenosis.

DIAGNOSIS.—The recognition of pulmonary stenosis is comparatively easy in the diagnosis of the congenital cardiac anomalies. Whenever the combination of cyanosis, systolic murmur, and cardiac

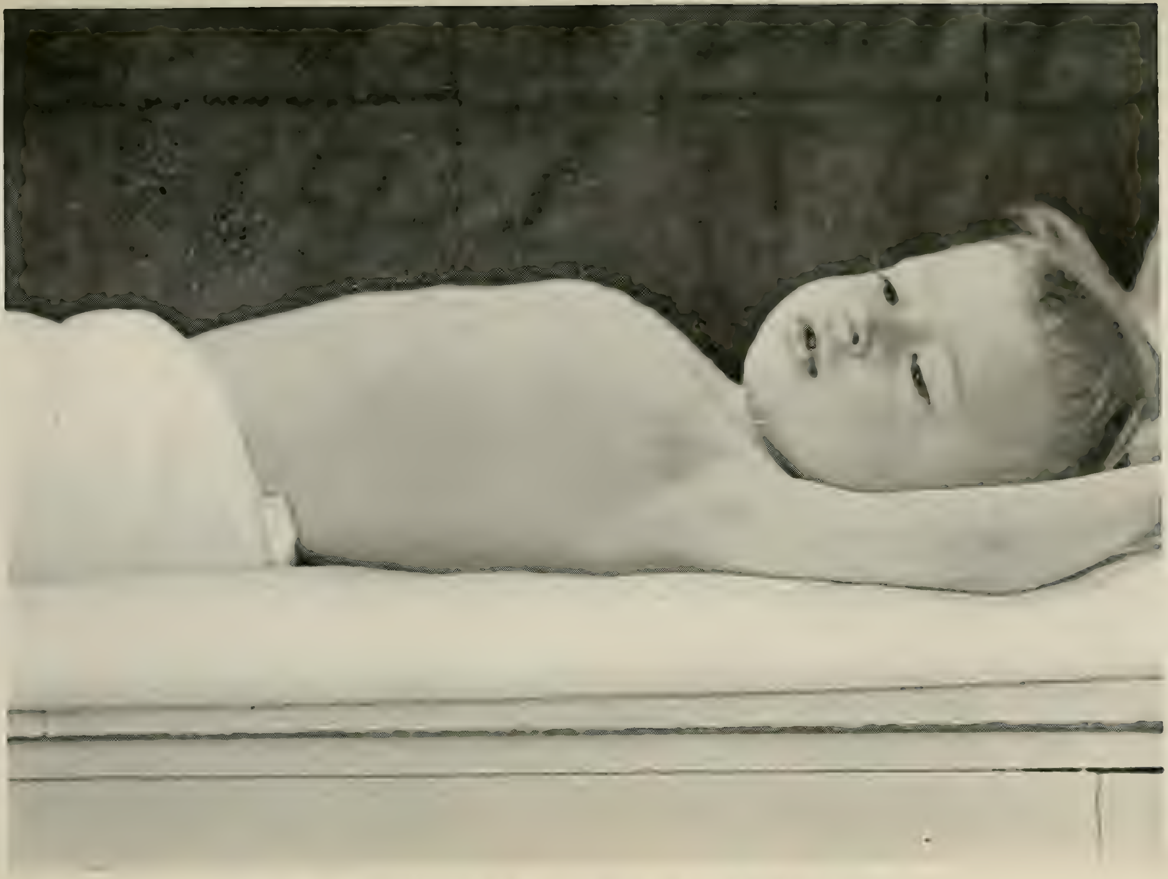


Fig. 215—Prominence of the Precordia in Congenital Cardiac Disease

enlargement is found, the physician may make a diagnosis of pulmonary stenosis with a fair degree of certainty that this lesion will be found post-mortem. The presence of a palpable systolic thrill still further confirms the diagnosis. The combination of " complete " transposition of the great vessels plus an opening in the interventricular septum may give the symptom complex of pulmonary stenosis without any real stenosis of the pulmonary artery, but this is a rare condition, and cannot usually be recognized by ordinary clinical methods.

In babies who die shortly after birth, the diagnosis must depend upon the association of cyanosis and murmur, evidences of cardiac enlargement not being present. In many of such cases the pulmonary stenosis is the sole lesion. If associated lesions are present, they cannot be recognized clinically. If in a baby dying shortly after birth, cyanosis is the only symptom, no murmur being present, the most probable lesion is complete atresia in some part of the pulmonary tract, although in such cases congenital atelectasis of the lung must be excluded. This last condition is recognized by the facts that asphyxia neonatorum is usually present at birth, that the cry is very feeble, that the respiration is very feeble and irregular, and that the cyanosis of the baby is lessened, not deepened by crying.

When a baby showing the characteristic symptom complex of pulmonary stenosis survives the early weeks, there is a possibility that this is the sole lesion, but it is much more probable that there is an associated compensating lesion. The signs of pulmonary stenosis so mask those of the associated lesion, that a positive recognition of the nature of the latter is usually impossible. Nevertheless, on a basis of probability, it is safest to assume that some additional lesion is present. If this be an open ductus arteriosus, the diagnosis can often be made from the fact that the murmur, instead of being only systolic, extends throughout the cardiac cycle,—the so-called " humming-top " murmur, or that the systolic murmur is transmitted into the carotid arteries. If no characteristic murmur be present, we must assume that the associated lesion is either a defect in the interventricular septum, or an open foramen ovale, or both. It is impossible to tell from the signs which of these lesions is present. In the order of probability they are first, both septum defects, second, interventricular septum defect, third, open foramen ovale.

The diagnosis of pulmonary stenosis may be summarized as follows:

1. When the combination of cyanosis, systolic murmur, and cardiac enlargement are present, pulmonary stenosis is usually present. A palpable systolic thrill makes the diagnosis almost certain.

2. If the baby dies shortly after birth, the probable diagnosis is pulmonary stenosis alone, or complete atresia with or without an associated lesion.

3. If the baby survives longer, the probable diagnosis is pulmonary stenosis with one or more associated lesions. If a " humming-top " murmur be present, or a murmur transmitted into the carotid arteries, the associated lesion is open ductus arteriosus. If no such murmur be present, the associated lesion is a defect in the interventricular septum, an open foramen ovale, or both.

4. If cyanosis and enlargement be present without a murmur, the probable diagnosis is complete atresia or general hypoplasia of the pulmonary artery, a rarer possibility being transposition of the great vessels.

PROGNOSIS.—The prognosis in a baby with congenital cyanosis from cardiac disease is grave at birth. Many cases only survive a few hours. If the baby survives the early weeks, the outlook as to the duration of life is entirely uncertain. The majority die in infancy or early childhood. A certain number live until late childhood, or early or even late adult life.

In those cases which survive the early weeks but die later, death usually does not occur from primary circulatory failure. The associated compensating lesion which is present in these cases appears to permit the circulation to be carried on without the mechanical obstruction which leads to progressive cardiac failure. The uncertainty of the prognosis comes from general lowering of the resistance of the child. These children seem to be particularly susceptible to infections involving the lungs, more so than to gastro-intestinal disturbance. Many cases, especially those reaching adult life, eventually develop tuberculosis. The usual cause of death is some intercurrent infection.

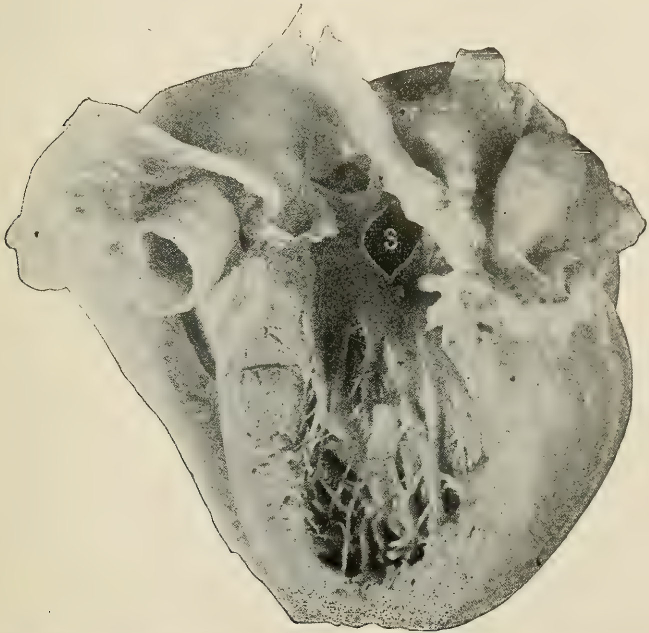
TREATMENT.—No special course of treatment is indicated in these cases. The feeding and hygienic care of the patient are very important. The only practical question which arises is how much limitation should be placed upon the normal activities of the growing child. Restriction of exercise is bad hygiene, and has an unfavorable effect upon general resistance. On the other hand, how great is the danger of undue strain upon the circulatory apparatus? The physician can only be guided by the symptoms produced by exertion. An amount of exercise which produces or increases dyspnea, which produces a feeling of weakness or a condition of exhaustion, or which causes any irregularity in the force and rhythm of the heart beat, should be avoided. With these limitations, the child should be brought up under the conditions which best conduce to normal development.

DEFECT OF THE INTERVENTRICULAR SEPTUM

This anomaly is much less common than stenosis of the pulmonary artery. It may occur as the only anomaly, but much more often it is associated with other lesions, particularly pulmonary stenosis and transposition of the great vessels.

PATHOLOGICAL ANATOMY.—In the majority of cases the defect is represented by a circular or oval opening of varying size, situated at the base of the interventricular septum just below the orifices of the great vessels. As rare anatomical curiosities are encountered cases in which the interventricular septum is completely absent (*cor biatriatrum*), or rudimentary.

FIG. 216



S, unclosed ventricular septum. Female 10 months old. Warren Museum, Harvard University

ETIOLOGY.—This lesion always represents a developmental anomaly occurring early in fetal life. In cases in which defect of the interventricular septum is the only lesion, it represents a primary failure of embryonic development, due to the general causes of developmental malformations. (See page 12). In the majority of cases, the defect occurs in association with pulmonary stenosis or transposition of the great vessels. The most satisfactory explanation of the frequent simultaneous occurrence of these lesions is that of Rokitansky, who traces them all to a deviation of the aortic septum. In defect of the interventricular septum the opening is caused by failure of the deviated aortic septum to unite properly with the interventricular septum. This may also be the cause of the cases which apparently represent a primary defect.

SYMPTOMS.—The most common symptoms of a "primary" defect of the interventricular septum are a systolic murmur with enlargement of the area of cardiac dulness. Cyanosis is almost invariably absent. In a few cases a transitory cyanosis on exertion has been observed.

The murmur in typical cases is systolic in time. It is very loud and harsh, but does not wholly replace the first heart sound. The murmur is loudest at the left border of the sternum at the second and third interspaces. It is also heard all over the precordia, and indeed, all over the chest. It is, however, *not transmitted into the carotid arteries*—an important point in diagnosis. The murmur has only been found absent in exceptional cases with a very large septum defect, and in such cases the diagnosis cannot be made. There is often with defective interventricular septum an accentuated pulmonic second sound. A palpable thrill has been found in some cases, but is usually absent.

The enlargement of the heart is due to a compensatory development of the right ventricle, which hypertrophies in the effort to maintain a normal pulmonary circulation in spite of the septum defect. The enlargement of the cardiac dulness is toward the right, but there is usually also some enlargement toward the left, with slight displacement of the apex beat. The evidences of cardiac enlargement do not appear for a considerable time, longer than in pulmonary stenosis. It may be years before enlargement of the area of precordial dulness can be definitely made out. This increases the difficulty of diagnosis, especially in infancy and early childhood. Enlargement always occurs eventually.

When a defect in the interventricular septum is the sole congenital cardiac anomaly, there are no other symptoms than the murmur and enlargement of the right heart. The general condition of the child, and its capacity for physical exertion are unaffected. Dyspnea and other symptoms of circulatory disturbance are only seen when pulmonary stenosis, or some rare congenital abnormality, is present in addition.

When the interventricular septum defect is "secondary," that is, occurring as an additional lesion in pulmonary stenosis, its symptoms are entirely masked by the murmur and cardiac enlargement which are characteristic of the latter disease.

DIAGNOSIS.—Defect of the interventricular septum is difficult to recognize clinically except in cases where it is the sole lesion. The diagnosis depends upon the combination of a loud, harsh systolic murmur and evidences of enlargement of the right ventricle. Pulmonary stenosis is excluded by the absence of cyanosis. When definite enlargement of the area of precordial dulness is present, patent ductus arteriosus as the sole lesion can be easily excluded, as this condition alone never gives cardiac enlargement. If the case be seen in early infancy, enlargement may be absent in interventricular septum defect. In such cases the differential diagnosis between septum defect and open ductus arteriosus is more difficult. It depends mainly upon the absence in septum defect, and the presence in open ductus, of transmission of the murmur into the carotid arteries. Of course, if the murmur be of the " humming-top " character, the lesion is open ductus.

When the defect of the interventricular septum is an accompaniment of other lesions, it cannot be recognized positively by clinical signs. If the presence of cyanosis points to pulmonary stenosis as the primary lesion in an infant who has survived the early weeks of life, we know that defect of the interventricular septum is the commonest secondary lesion, and may assume that it is probably present. The combination of interventricular septum defect and open ductus arteriosus is a very rare one. It can only be recognized by the concurrence of distinct evidences of cardiac enlargement and a murmur transmitted into the carotids, or of the " humming-top " variety. If no enlargement be present, only the open ductus can be recognized, and the septum defect will be overlooked.

In older children, in whom the presence of a cardiac lesion in infancy has been overlooked, the differentiation of congenital septum defect from the mitral insufficiency of acquired cardiac disease is sometimes extremely difficult. A history of a rheumatic fever attack, or of any subjective symptoms suggesting cardiac disease, points toward acquired endocarditis. The absence of cardiac or rheumatic symptoms, the harsh character of the murmur, and the fact that it is usually louder at the base of the heart than at the apex, point toward congenital defect of the interventricular septum.

PROGNOSIS.—The prognosis of uncomplicated defect of the interventricular septum is good in so far as life and normal development are concerned. This lesion does not appear either to embarrass the circulation, or to diminish the physical resistance toward adverse influences. The lesion persists throughout life, without clinical significance. When the septum defect is associated with some other lesion, the prognosis is that of the associated lesion.

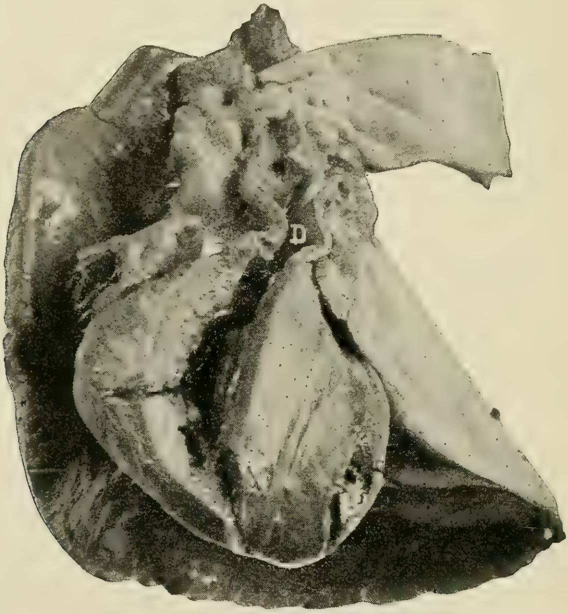
TREATMENT.—None is required in uncomplicated cases. When the lesion is associated with pulmonary stenosis, the treatment is that of the latter disease.

PATENT DUCTUS ARTERIOSUS BOTALLI

If post-mortem records be taken as a criterion, this lesion is much less common than either pulmonary stenosis or defect of the interventricular septum. I am convinced, however, from clinical experience, that open ductus arteriosus is not necessarily a permanent lesion, and that in many cases in which all the clinical evidences of its existence are present, complete recovery takes place. As a result, I believe that the lesion is far more common than is generally stated in the literature.

PATHOLOGICAL ANATOMY.—The lesion represents the persistence of a condition which is normal only in fetal life. The normal closure of the ductus through thrombus organization and obliterating endarteritis does not take place. Instead of an impervious fibrous cord or a bit of fibrous tissue connecting the pulmonary artery with the beginning of the descending aorta, there is an open blood vessel. The ductus may be found widely patulous, lined with normal endothelium, or there may be evidences of a certain amount of endarteritis, causing a narrowing of the open lumen.

FIG. 217



D, open ductus arteriosus. Male, 16 days old. Warren Museum, Harvard University

ETIOLOGY.—The lesion is developmental in origin. I believe that it represents, not always *arrested development*, but often merely *delayed development*. My reasons for this belief are, first, that I have seen at autopsy in infants in the second year, evidences of a recent obliterating endarteritis in open ductus arteriosus; second, that I have seen a number of cases showing the typical clinical picture of this lesion, in which all the signs disappeared in later life.

The cause of the disturbance of development is not definitely known. When the lesion occurs in association with pulmonary stenosis, the cause is probably the embarrassment of the circulation produced by that lesion, and the open ductus arteriosus is secondary, compensatory and usually permanent. When open ductus arteriosus is the sole lesion, I believe the causes are those of retarded normal development in general, which in this instance affects the circulatory system. The lesion is then primary, and usually not permanent.

SYMPTOMS.—The only notable clinical manifestation is the presence of a cardiac murmur. The murmur is usually either systolic in time, or is heard throughout the cardiac cycle. In rare cases, a diastolic murmur only has been heard. When the murmur extends throughout the cardiac cycle, a continuous humming or whirring sound is heard, to which the term " humming-top " murmur has been applied. This murmur is not heard in the majority of cases of open ductus arteriosus, but when present it is diagnostic.

The systolic murmur usually heard is not so rough and harsh as the murmurs of pulmonary stenosis and interventricular septum defect. It often much resembles in sound the murmur of mitral insufficiency. It is usually heard loudest over the second and third left interspaces near the left border of the sternum. It is also heard all over the precordia, is transmitted into the axilla, and may be heard all over the back of the chest. The most important feature in the diagnosis of open ductus arteriosus is that *the murmur*, whether merely systolic or "humming-top," *is always transmitted loudly into the carotid arteries.*

Enlargement of the area of cardiac dulness is not present in uncomplicated cases. There may be, however, in long-standing cases, an abnormal area of dulness, above and distinct from the cardiac dulness, in the first and second left interspaces (Gerhardt's sign). This is due to a dilatation of the pulmonary artery, which only takes place late in the course of the condition. This dilatation may be visible in roentgenograms. Cyanosis is practically always absent. Occasionally a palpable systolic thrill has been observed in uncomplicated cases.

Patent ductus arteriosus has little or no effect upon normal development and resistance. In long-standing cases a tendency toward infection of the respiratory tract has been observed, but a causal connection is not very clear. In many cases the murmur disappears in the course of time. In other cases it persists throughout life.

DIAGNOSIS.—In cases in which a "humming-top" murmur is heard, the diagnosis of open ductus arteriosus can be made with a fair degree of certainty. In cases in which the murmur is systolic in time, the diagnosis of open ductus depends upon transmission of the murmur into the carotid arteries. If a murmur with such transmission be present without either cyanosis or enlargement of the precordial dulness, the diagnosis is open ductus arteriosus alone. If with a murmur characteristic of open ductus, there be cardiac enlargement without cyanosis, the diagnosis is open ductus arteriosus plus defect in the interventricular septum. If cyanosis be present, the additional lesion is pulmonary stenosis.

PROGNOSIS.—In cases showing the evidences of open ductus arteriosus as the sole lesion, the prognosis is favorable. It is generally stated in the literature to be favorable as to the duration of life, but not favorable as to disappearance of the lesion. This is because the statement is based on the age at death of patients in whom the lesion was found post-mortem. I believe that when open ductus arteriosus is diagnosed as the sole lesion in the first two or three years of life, the prognosis is also favorable as to complete recovery. I have seen a number of such cases in which the physical signs of the lesion disappeared completely in later life, and I believe that it represented only retarded development, a delay in the normal process of closure by obliterating endarteritis.

TREATMENT.—No special treatment is indicated.

TRANSPOSITION OF THE GREAT VESSELS

This lesion is rare in comparison with those described above.

PATHOLOGICAL ANATOMY.—Normally the pulmonary artery arises in front and to the right, passing immediately behind the aorta which arises behind and to the left. There are two forms of transposition. In both, the point of origin of the aorta is in front to the right, that of the pulmonary artery being behind to the left. In one form, in spite of the change of position, each vessel opens from its proper ventricle. In the second form the aorta opens from the right ventricle, the pulmonary artery from the left. In the majority of cases transposition of the great vessels is associated with some form of stenosis of the pulmonary artery, and usually there is also a defect in the interventricular septum.

ETIOLOGY.—The most plausible explanation of the lesion is that of Rokitansky. In the conus arteriosus which forms the upper part of the primitive aorta, the formation of the transverse aortic septum places the lumen of the aorta in front. The torsion which brings the opening of the aorta behind is due to a kinking in the bulbus cordis, which forms the lower part of the primitive aorta and pulmonary artery. This torsion is represented by the spiral arrangement of the septum in the bulbus cordis. If this normal kinking and

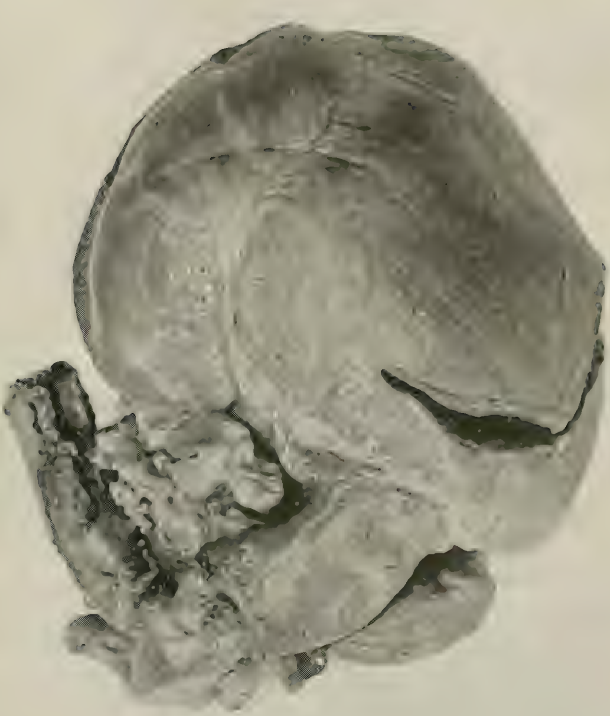


FIG. 218—Complete Transposition of the Great Vessels

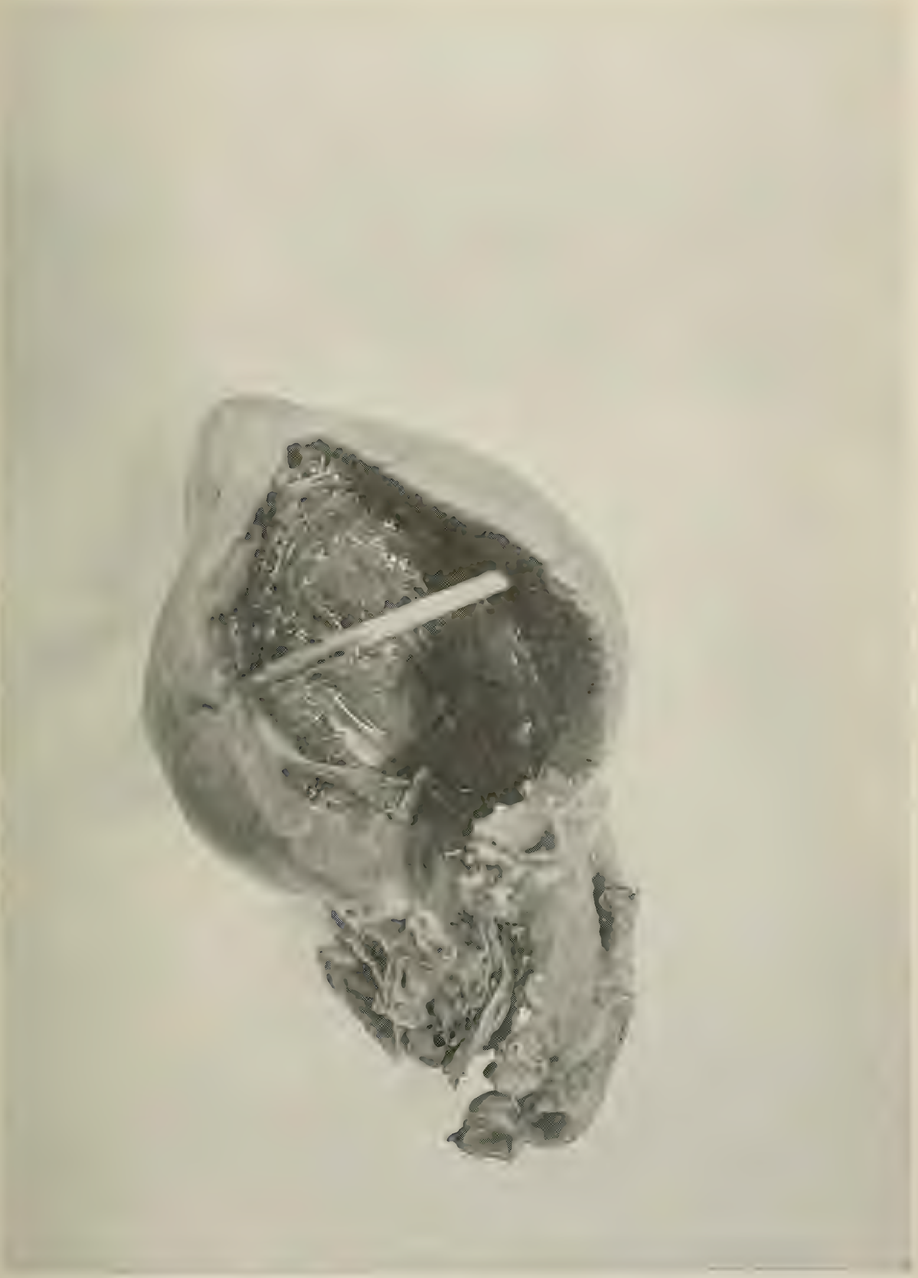


Fig. 219—Complete Transposition of the Great Vessels with Defective Interventricular Septum

torsion does not occur, or is slightly reversed, the aorta will arise in front and to the right, the pulmonary artery behind and to the left. In the first form of transposition there is a sympathetic adjustment of the interventricular septum in its union with the aortopulmonary septum, which causes each great vessel to open into its proper ventricle. This form is called by Rokitansky "corrected" transposition. In the second form the interventricular septum unites with the malposed aortopulmonary septum without sympathetic adjustment, causing the aorta to open from the right ventricle and the pulmonary artery from the left ventricle. This is called "complete" transposition.

SYMPTOMS.—Corrected transposition may occur as the sole lesion, and has no recognizable symptoms. When associated with other lesions, only the symptoms of the associated anomalies are found.

Complete transposition has occurred as the sole lesion, and has been correctly diagnosed during life. The symptoms are cyanosis, without murmur or evidences of cardiac enlargement. When associated with other lesions, there are no signs characteristic of transposition.

DIAGNOSIS.—Corrected transposition cannot be recognized clinically. In the most common combination, that of complete or corrected transposition with pulmonary stenosis and defective interventricular septum, only the last two can be diagnosed. In a case showing persistent congenital cyanosis, without murmur, and without cardiac enlargement, complete transposition is the most probable diagnosis. If with cyanosis but no murmur, cardiac enlargement is also present, complete pulmonary artery hypoplasia is more probable.

PROGNOSIS.—This depends on the associated lesions, and is usually that of pulmonary stenosis.

TREATMENT.—The treatment is that of pulmonary stenosis.

DIFFERENTIAL DIAGNOSIS OF THE COMMONER CONGENITAL CARDIAC ANOMALIES

The difficulties in the differential diagnosis of the congenital cardiac diseases are increased by two facts: First, exceptions to the general rules upon which the diagnosis is based are encountered at times; second, rare anomalies are encountered showing the same signs as the commoner lesions. If this be borne in mind, the table on page 30 may prove useful for reference:

Subject to the exceptions noted above, the following rules are useful:

1. A case showing cyanosis and murmur with enlargement of the cardiac dulness or palpable thrill, or both, is one of pulmonary stenosis. If the baby dies shortly after birth, the most probable

TABLE 63

Differential Diagnosis and Prognosis of the Commoner Congenital Cardiac Anomalies

	MURMUR		CYANOSIS	ENLARGEMENT	THRILL	PROGNOSIS		
	TIME	TRANSMISSION TO CAROTIDS				EARLY FATAL ENDING	LATER FATAL ENDING	FAVORABLE
Pulmonary Stenosis alone................	Systolic	○	+	+	+(occ.○)	+	occ.	rare
Defective Interventricular Septum alone ..	Systolic	○	○	+	○(occ.+)	○	rare	+
Open Ductus Arteriosus alone............	Systolic or "humming-top"	+	○	○	○(occ.+)	○	rare	+
Pulmonary Stenosis and Defective Septum	Systolic	○	+	+	+(occ.○)	○	+	rare
Pulmonary Stenosis and Open Ductus.....	Systolic or "humming-top"	+	+	+	+(occ.○)	○	+	rare
Defective Septum and Open Ductus......	Systolic or "humming-top"	+	○	+	○(occ.+)	○	occ.	+
Transposition of Great Vessels alone......	○	○	+	○	○	○	+	rare

lesion is pulmonary stenosis alone, or complete pulmonary atresia with other lesions. If the baby survives early infancy, or lives on into childhood, the pulmonary stenosis is probably not complete, and is associated with some other lesion. If the murmur is notably transmitted into the vessels of the neck, or if a " humming-top " murmur is present, the additional lesion is probably open ductus arteriosus. If the murmur has neither of these characteristics, the complicating lesion is probably defective interventricular septum.

2. A case showing a murmur and enlargement, without cyanosis, is probably defective interventricular septum. If the murmur is not transmitted into the vessels of the neck, this lesion exists alone. If the murmur is so transmitted, or if the " humming-top " murmur is present, the lesion is probably combined with open ductus arteriosus.

3. A case showing a murmur, without either cyanosis or enlargement, especially if the murmur is markedly transmitted into the vessels of the neck, or if it extends into diastole, is probably one of open ductus arteriosus alone. If the murmur is of the " humming-top " variety, extending throughout the cardiac cycle, the diagnosis of this lesion is almost certain.

PROBLEMS AND RESEARCH

The aspects of practical importance in connection with congenital cardiac disease are those of diagnosis and prognosis. Of the great variety of possible cardiac anomalies, the majority are so rare, that it is unlikely that we shall ever be able to establish a satisfactory basis for their diagnosis. As to the commoner anomalies, it seems as if we had reached the limit of progress in diagnosis based on the more ordinary methods of examination. In the effort to establish a basis of diagnosis, attention has been directed almost exclusively to the cases which finally came to autopsy. The following throughout life of a series of cases showing clinical evidences of congenital disease of the heart, would probably supply a very valuable addition to our knowledge of the prognosis of these affections.

The principal question now awaiting answer in connection with congenital cardiac disease, is what light will be thrown upon these affections by the use of certain of the newer diagnostic procedures based on physiological methods. The most important of these newer methods of investigation is that with the electrocardiograph. The electrocardiographic curve in congenital cardiac disease has been studied by a number of observers. So far, very few positive points have been brought out, and these do not seem to be of any very immediate practical value. The principal points elicited in some cases are the following: 1. A deep exaggeration of the S wave in lead I. This is only a corroborative sign of marked right-sided cardiac hypertrophy; 2. an extreme amplitude of the curves in several leads, considered by Lewis a valuable sign of congenital valvular or septal defects; 3. a transposed electrocardiogram is the most valuable sign which we possess of true (mirror-picture) dextrocardia.

Roentgen-ray examination has in certain cases shown dilatation or hypoplasia of the great vessels. In the majority of cases, however, it is only of value in corroborating the clinical finding of hypertrophy of the right ventricle.

The estimation of the oxygen content of the alveolar air is considered by Plesch an important diagnostic point between defects due to abnormal communication between the right and left sides of the heart, and those due to obstruction of the pulmonary artery. In septal defects and similar conditions the oxygen content is raised.

II. INFECTIONS

The diseases of the heart due to bacterial infection form the largest group in early life. The heart of the child appears to be particularly susceptible to invasion with pathogenic microörganisms, and acute infection is relatively more common in children than in adults. The diseases in which the patient suffers from the immediate effects of bacterial invasion are described under the term *acute*. The diseases in which the patient suffers from the after effects of the damage done by a preceding infection, are described under the term *chronic*.

It has long been customary to subdivide the acute infections of the heart on the basis of the part of the heart affected. Under this method of classification, we have acute endocarditis, acute myocarditis, and acute pericarditis. In this connection it should be remembered that in children acute infection of a particular part of the heart alone is very rare. The endocardium and the pericardium are respectively very rarely the seat of acute infection without a coincident involvement of the myocardium. This is particularly true of the rheumatic fever infection, which forms the largest etiological group encountered in early life. The most common finding in fatal cases of this disease in childhood is acute infection of all three of the cardiac tissues, which would best be described by the term acute carditis. The clinical symptoms of acute infection of the heart differ somewhat according to the part of the heart involved, and it is for this reason that acute endocarditis and acute pericarditis will be described separately. Acute myocarditis never occurs alone, always accompanies acute endo- and pericarditis in rheumatic fever infection, and has no signs peculiar to itself. For this reason it will not be described as a separate disease. The same is true of chronic myocarditis.

ACUTE ENDOCARDITIS

This is an acute inflammation of the lining membrane of the heart and its valves, caused by bacterial infection.

ETIOLOGY. The Microörganisms.—There is no variety of bacteria exclusively concerned in the production of acute endocarditis. The streptococcus, staphylococcus, pneumococcus, and gonococcus are the forms which have been most frequently found. Less frequently, the tubercle bacillus, the typhoid bacillus, the anthrax bacillus, the colon bacillus, the diphtheria bacillus, the bacillus pyocyaneus, and bacillus of influenza have been reported.

In the great majority of cases of acute endocarditis in children, the lesion occurs as a manifestation of rheumatic fever infection. The particular microörganism which causes rheumatic fever has never been positively determined. (See Vol. II. page 320). The weight of modern opinion inclines toward the view that it is a certain strain or certain strains of streptococcus, having particular selective affinities.

The fact that acute endocarditis is almost always an accompaniment of some recognizable type of infection, has led to the common statement that the disease is not a primary infection, but usually secondary. This statement is based on the conditions which obtain in adults, in whom rheumatic fever is regarded as primarily a disease of the joints, the endocarditis being regarded as a secondary complication. In children rheumatic fever cannot be regarded as primarily a disease of the joints, but as one in which arthritis and endocarditis are coördinate manifestations, the latter being of an importance equal to if not greater than the former. Therefore the acute endocarditis of rheumatic fever should be regarded as a primary manifestation of the disease. In children acute endocarditis is frequently a clinically primary manifestation of rheumatic fever, as cases occur in which there is no evidence of present or previous arthritis, but only of acute endocarditis, which cases have proved to be instances of rheumatic fever infection through the subsequent appearance of arthritis. The primary character of the rheumatic fever infection is probably due to a selective affinity of the rheumatic fever microörganism for the endocardium. This theory is borne out by the fact that in general the experimental injection of microörganisms into the blood does not result in endocarditis unless the valves of the heart have been injured in some way. Only in experimental injections with the "micrococcus rheumaticus," or with streptococci derived from rheumatic fever patients, has endocarditis been produced without previous valve injury. (Poynton, Paine, Cole, Rosenow.)

It is only in rheumatic fever that endocarditis may be considered primary. In the small group of cases of acute endocarditis not due to rheumatic fever, the process is always secondary to some infectious process localized elsewhere.

In this group the acute endocarditis is merely an incident in septicemia. A condition of bacteriemia is not uncommon in most of the recognized infectious diseases, and when in certain cases the microörganisms circulating in the blood are deposited and develop upon the valves of the heart, acute endocarditis is seen as a complication. This may occur in any infection. The microörganism may be the one acting as the cause of the disease, as in pneumonia or gonorrhea or it may be a secondary invader as in scarlet fever and measles. The disease in which secondary endocarditis occurs most frequently is scarlet fever, but it may rarely occur in children in any of the common infections.

There are a few further etiological differences in the acute endocarditis of children as distinguished from that of adults. In most descriptions of the disease an anatomical distinction is drawn between verrucose and ulcerative forms, and a clinical distinction between benign and malignant forms. In children these distinctions have no etiological basis. The lesions characteristic of ulcerative endocarditis are rare in children, but are seen both in rheumatic fever and in secondary infections complicating other diseases. Cases whose clinical severity is described by the term malignant, are also seen in both etiologic types. The clinical severity of the case, as well as the ulcerative or verrucose character of the lesion depends, not upon the variety of the infecting microörganism, nor upon its numbers in the blood, but upon the virulence of the particular bacterial strain, and possibly also upon the resistance of the patient. The particular type of ulcerative or malignant endocarditis, comparatively common in adults, in which the lesion is part of a general septicopyemia originating in a local septic process, is rare in early life.

There is a type of infectious endocarditis which has been described under the names *endocarditis lenta*, or *subacute endocarditis*, which is now regarded by many writers as a specific infectious disease, due to the *streptococcus viridans* of Schottmüller. A number of cases of this type have been collected by Libman. Most of them were in adults. Apparently they showed not only a characteristic clinical picture, but also a characteristic pathological anatomy. The same clinical picture is seen in children in processes which certainly represent common types of rheumatic fever in early life. Whether some of these cases are due to the streptococcus viridans and have characteristic lesions, is yet to be determined.

PORTALS OF ENTRY.—The portals of entry in rheumatic fever endocarditis have been discussed under the description of that disease. (Vol. II, p. 669.) The evidence points to the tonsils as the usual portal of entry. In the cases secondary to some other form of infection, the organisms may enter through the tonsils, mouth, around the teeth, through other parts of the alimentary canal, through the genito-urinary tract, through the respiratory tract, and through the skin. The portal of entry varies with the type of infection to which the endocarditis is secondary.

PREDISPOSITION.—The factors concerned in predisposition toward the rheumatic fever infection have been discussed under that disease. Acute endocarditis is rare in infancy, and is uncommon before the fifth year. From this age it occurs with increasing frequency throughout childhood. In rheumatic fever, constitutional predisposition undoubtedly plays an important part. In secondary forms, constitutional factors are less important. Strain and tension on the heart valves may play a part, but the chief factor is the virulence of the infecting organism.

PATHOLOGICAL ANATOMY.—When the lesions of endocarditis are found in children, the connective tissue and the basement substance are principally concerned in the inflammatory process. The endocardium which forms the valves is that which is most frequently inflamed, but other portions of it are by no means exempt. In some cases there is swelling of the valves, which are thickened, their surfaces remaining smooth, the basement substance is swollen, and there is a moderate production of new connective-tissue cells. In other cases the growth of connective-tissue cells is very much more marked, the basement substance is broken up, and the little cellular fungus-masses, called *vegetations*, project from the free surface of the endocardium (verrucous endocarditis).

On the surface of these vegetations thrombi may be formed, and bacteria are sometimes present. In still other cases the cellular growth in some places forms vegetations, and in others degenerates, and thus portions of the valves are destroyed. This is *acute ulcerative endocarditis*. In the simple form of acute endocarditis emboli may occur, which from their size usually produce mechanical disturbances alone, while in the malignant form the emboli are usually smaller, and, being septic, produce inflammatory rather than mechanical disturbance, giving rise to miliary abscesses in various parts of the body, such as the liver, kidneys, spleen, stomach, intestine, brain, eye, joints, and skin.

SYMPTOMS.—The symptoms of acute endocarditis are usually described under two headings, simple endocarditis, and malignant endocarditis. The former is the usual type seen in children, the latter being rare.

COMMON ACUTE ENDOCARDITIS.—The symptoms differ somewhat according to whether the disease represents a manifestation of rheumatic fever, or a complication of some other infection. When the disease arises in connection with some other disease, the symptoms are especially likely to be masked by those of the disease which it complicates. In most cases the endocarditis develops insidiously without any additional symptoms such as pain or precordial distress, and its presence is not recognized until a careful examination of the heart reveals a murmur; in some cases, pronounced and even violent cardiac symptoms are present from the beginning. In rare cases the first evidence of an endocarditis may come from symptoms of embolism such as hemiplegia, hematuria, dyspnea, cough or localized pain, as in the thorax or abdomen. If the muscular tissue is involved as well as the endocardium, the general cardiac symptoms of dyspnea, cyanosis, and palpitation are still more marked.

When acute endocarditis is a manifestation of rheumatic fever, the principal symptoms are fever, signs of cardiac weakness, and an endocardial murmur. As far as the heart is concerned, the symptoms are the same whether the endocarditis is the only manifestation of rheumatic fever present, or whether there are also evidences of arthritis.

Fever is invariably present in acute endocarditis, and vice versa, the presence of fever always means that the process in the heart is an actually present acute infection. The characteristics of the fever curve have been described in detail under rheumatic fever. It is irregular, often remittent, and the degree of temperature is very variable.

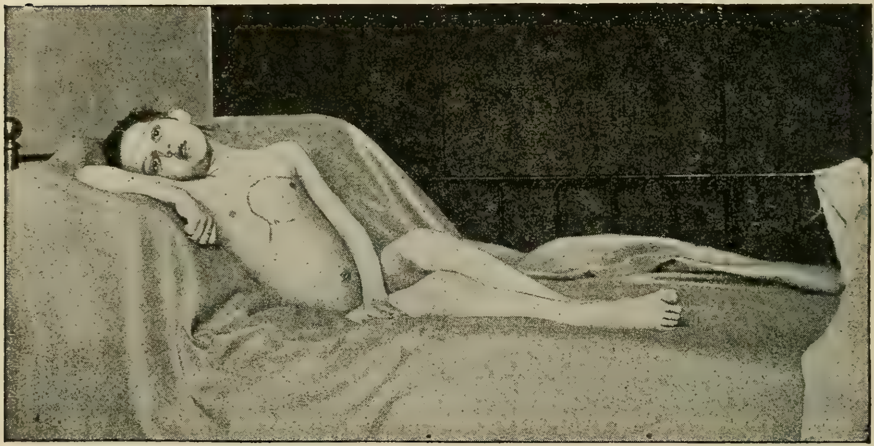
In the adult the development of acute endocarditis in the course of an attack of rheumatic fever is usually insidious, betraying itself only by the appearance of an endocardial murmur. In children the development of acute endocarditis usually, but not always, manifests itself by the presence of symptoms referable to the heart. These symptoms are not due to any mechanical obstruction of the circulation through the development of vegetations upon the valves, but are due to the acute myocarditis which almost invariably accompanies the acute endocarditis of rheumatic fever, and dilatation of the heart is produced not by overload, but by the weakening of the myocardium as a result of infection. The symptoms vary very greatly in severity in different cases. In general they are those of cardiac weakness. In the mildest cases the principal symptom is an increased rapidity of the heart's action, with certain peculiarities of the impulse and heart sounds, which are hard to describe, but easy to recognize. The impulse gives a staccato impression, seeming forcible, but ill-sustained. The first heart sound is louder and shorter than normal. The term "irritable" best describes this type of cardiac action. There may also be slight irregularity of rhythm.

The most common symptoms of acute endocarditis in children are palpitation and precordial pain. In many cases, irritable heart, precordial pain, and palpitation are the only symptoms. In more severe cases dyspnea is present in addition, and may amount to orthopnea. Cough, often severe and racking, is a very common symptom, and in children is usually the first sign of the passive congestion produced by weakened heart action. The next sign to appear is enlargement and tenderness of the liver.

These are the usual symptoms of the early stage of cases of moderate severity. The pulse does not feel particularly weak, and does not show any marked irregularity, except an occasional intermitting. Edema is usually absent in this stage of the disease, even though dyspnea and cough be quite marked. Exceptionally severe cases may die in this stage of the disease, without the appearance of edema of the skin. The unfavorable features which precede a fatal ending are the sudden appearance of obstinate vomiting, and a rapid diminution in the quantity of the urine excreted. The character of the radial pulse is very deceptive in these cases, remaining comparatively strong up to the end. The heart sounds, however, become weak, with more and more frequent intermitting of the heart action, until death occurs suddenly.

In more prolonged cases of the severer type the progress is more gradual. The patient's condition varies from day to day. Sometimes cough, dyspnea, and precordial distress are less, sometimes greater. The fever continues. There is, however, a gradual diminution in the quantity of urine excreted. Eventually edema develops, beginning in the feet, ankles, or legs. At any time, the course of the disease may take a favorable turn, edema may disappear, the temperature may fall, and the cough, dyspnea and precordial distress may lessen. In some cases edema may become general, and fluid may appear in the peritoneal or pleural cavities. There is always still the possibility of rally. If, however, obstinate vomiting sets in, the outcome is usually unfavorable.

FIG. 220



Acute endocarditis. Mitral insufficiency. Lack of compensation. Orthopnoea. Female, 9 years old

It appears, then, that the clinical picture presented by acute endocarditis in childhood is often the same as that with which we associate the term " broken compensation " in adults. I have often heard the term " broken compensation " applied to cases in children showing the symptoms described above, especially in children having had previous evidences of a chronic valvular lesion. I wish strongly to deprecate the use of this term as applied to the symptoms of cardiac weakness when they occur as manifestations of acute endocarditis. The term broken compensation implies that the cause of the cardiac insufficiency is overstrain. The theory is that the valvular lesions of a permanently damaged heart are compensated by cardiac hypertrophy, and that the symptoms of precordial pain, palpitation, cough, dyspnea, orthopnea, enlarged liver, and edema are produced by an excessive strain thrown on the heart by overexertion or some similar cause. This is the true explanation of the occurrence of these symptoms in most cases of valvular disease in adults, and of the cases in children in which the symptoms occur in chronic endocarditis, without fever or other evidence of a fresh infection. In early life, however, such cases form the minority of those showing symptoms of cardiac insufficiency. The majority show fever, and the cardiac symptoms are a manifestation of an acute endocarditis, caused by a fresh infection localized in the heart,—which is the commonest cause in childhood of the symptoms and signs of weakness of the heart.

The other symptoms, not immediately connected with the heart, characteristic of rheumatic fever, are usually present. The most notable is the rapidly progressing anemia. There is usually a moderate leukocytosis. The urine is diminished, the quantity excreted varying with the severity of the cardiac involvement. A slight trace of albumin, with a few casts and blood corpuscles, is usually present, the urine showing the characteristics of passive congestion. A careful record should always be kept of the daily quantity of urine, as this is the most important prognostic indication and therapeutic guide.

PHYSICAL EXAMINATION.—The cardiac impulse is visible and palpable, and usually fairly forcible. It is notably diffuse, often affecting the entire precordia, and often showing an undulatory appearance. In the early stages of a first attack of acute endocarditis, the area of precordial dulness is not notably increased. In the course of the disease, a moderate enlargement gradually occurs. In cases in which a previous attack has left behind a chronic valvular lesion, the area of precordial dulness is markedly enlarged from the beginning of the new acute attack.

The heart sounds are not particularly faint, but the first sound is shortened. There may be occasional irregular intermitting. In a first attack, an endocardial murmur may not be heard for the first few days, but soon develops. If there has been a previous attack, an endocardial murmur is usually present from the start. The murmurs need no detailed description, differing in no way from those heard in adults. The character of the murmur differs with the valvular lesion. The systolic murmur of mitral insufficiency is by far the most common in children, especially in acute endocarditis. Diastolic and presystolic murmurs are sometimes heard especially in cases in which there has been a previous endocarditis. A more detailed description of the endocardial murmurs of childhood will be given under chronic endocarditis. The pulmonic second sound is usually markedly accentuated. Pulsation of the vessels of the neck is often present.

Other points to be especially noted on physical examination are the amount of dyspnea, the condition of the lungs, the condition of the liver, and the presence or absence of edema. The breathing is often not accelerated, but is labored, with notable contraction of the accessory muscles of respiration, and dilatation of the alae nasi. Sometimes only the subjective sensation of oppressed breathing is present, without signs noticeable on inspection. In severe cases the breathing is rapid as well as labored. The dyspnea may amount to orthopnea. Examination of the lungs is usually negative, but severe cases may show diffuse moist râles, or evidences of fluid in the pleural cavities. The liver is enlarged and tender in severe cases. Edema usually appears first in the feet and ankles, may become general, and ascites is occasionally seen.

MALIGNANT ENDOCARDITIS.—The symptoms of malignant endocarditis are necessarily varied and indefinite, arising as they usually do in the course of some other disease with simply an exacerbation of the previous symptoms, especially of the temperature. There are usually the general symptoms of intermittent temperature, sweating, delirium, and failure in strength. Two types of the disease, septic or pyemic, and typhoidal, are recognized. The *septic type* is characterized by the signs of septic infection, rigors, sweating, and irregular temperature, while the *typhoidal type* shows diarrhea, dry tongue, distended and tympanitic abdomen, enlargement of the spleen, profuse sweating, and a petechial efflorescence on the skin. A clear distinction between the two forms, however, is often not marked. Cerebral symptoms may arise, and simulate meningitis or malaria. Embolic processes may produce special symptoms, such as paralysis, coma, bloody urine, retinal hemorrhage, peritonitis from infarction of the spleen, and in some cases pulmonary symptoms simulating pneumonia. The duration of the attack depends mostly on the disease to which it is secondary, and may vary from a few days to as many weeks. A marked leucocytosis is present.

DIAGNOSIS.—The diagnosis of the simple acute endocarditis which occurs as a secondary complication in many acute infections is very difficult. The only sign is the development of an endocardial murmur with or without symptoms of cardiac weakness. This does not, however, necessarily mean that there is a valvular lesion. A murmur due to *relative* mitral insufficiency and signs of cardiac weakness, may be produced by the dilatation which occurs as a result of the action of the toxin of the acute infection upon the myocardium, and this is the commonest cause of these signs in the course of an acute infection. Of course, if a presystolic or diastolic murmur develops, acute endocarditis is present, but such murmurs are rare. In most cases, endocarditis can only be positively recognized by the persistence of the murmur after recovery from the primary infection.

In cases of acute endocarditis occurring as a manifestation of rheumatic fever infection the diagnosis is comparatively easy. The presence of fever without any recognizable cause outside the heart, with an endocardial murmur, and often with cardiac symptoms, presents a clinical picture which is very characteristic. The physician easily recognizes the presence of a valvular lesion. He may be in doubt whether any cardiac symptoms which may be present are due to the overstrain of a previously damaged heart, or to a fresh infection of the endocardium. The point which determines the diagnosis of an *acute* endocarditis is the presence of fever without other cause.

The diagnosis of acute endocarditis may be made under the following conditions:—

1. When an endocardial murmur develops in the course of a rheumatic fever attack characterized by arthritis.
2. When the combination of fever, endocardial murmur, and symptoms of cardiac weakness is present, without other cause for the fever.
3. When fever without other cause develops in the course of a case of chronic endocarditis.
4. When signs of cardiac weakness accompanied by fever develop in the course of a case of chronic endocarditis.

The history of a previous attack of rheumatic fever, whether the manifestations have been arthritic, cardiac, or choreic, is a strong evidence that a febrile attack without other cause is due to acute endocarditis.

Malignant endocarditis is differentiated from the common form by the graver constitutional signs, the higher temperature, and the septic or typhoidal symptoms. From typhoid fever it is to be distinguished by its abrupt onset, irregular temperature, precordial pain and distress, marked leukocytosis, and negative Widal reaction. Malaria can be eliminated by the absence of the plasmodium malariae and the presence of leukocytosis.

COURSE AND PROGNOSIS.—The duration of an attack of acute endocarditis due to rheumatic fever is extremely variable. It may be less than a week, or it may be many weeks. No rule can be given, but the greater number of cases usually last for two or three weeks. The severity of the symptoms also is apt to vary from time to time in the course of the attack. Patients often begin to show improvement and then get worse again. Death may occur at any time in the course of the disease, either from a general exhaustion, or, more often, from sudden heart failure.

The best measure of the progress of the case is the daily quantity of urine. This rises when the patient improves, and falls when he grows worse. A persistent and progressive fall in the quantity of urine is of bad prognostic import. The fever curve is of little value in estimating the course of the disease, as it depends as much on the course of the infection in the endocardium as in the myocardium, but it is the course in the latter which influences prognosis. A permanent return of the temperature to the normal is a favorable sign, but it must be remembered that *relapses are very common.* The severity of the other signs of cardiac weakness, especially dyspnea, enlargement of the liver, and edema, are also a valuable measure of the progress of the case. The appearance of edema is a serious, but not necessarily fatal, sign. The appearance of vomiting is a very bad sign, and often means that a fatal ending is not far off. The character of the valvular lesions present is not of much prognostic significance, except that the prognosis is rather more grave in cases showing evidence of involvement of the aortic valves.

The mortality of acute endocarditis in childhood is higher than is generally recognized. In many cases the cause of death is considered to be broken compensation in chronic endocarditis, when the real cause is a fresh acute infection. In a series of hospital cases, the mortality from acute infection of the heart, including both endocarditis and pericarditis, was 55 out of 264 cases, or 20 per cent. The mortality from chronic endocarditis was 10 out of 58 cases, or 17 per cent. The mortality from acute endocarditis without pericarditis was 27 out of 148 cases, or 12 per cent. Of course hospital mortality figures are always likely to be somewhat above those of private practice. But these figures refer only to the immediate mortality in the particular attack. The tendency of rheumatic fever infection in childhood toward recurrent attacks of acute cardiac infection gravely affects the prognosis in the cases which recover from their first attack of acute endocarditis.

There is a possibility that complete recovery may occur after an attack of acute endocarditis from rheumatic fever, but it is hardly more than a possibility. *In the great majority of cases the persistence of the endocardial murmur shows that the heart is permanently damaged.* What is the outlook in these cases? They are still in danger from two causes which are likely to develop in the future. One is the liability to a recurrent attack of rheumatic fever, with a fresh, acute endocarditis, with all its uncertainties of prognosis. This is the chief danger in acute endocarditis acquired in childhood. The other danger comes from the liability to broken compensation from overexertion. This is a less important danger in childhood, and will be discussed under chronic endocarditis. In my series of cases, 209 children were discharged from the hospital with evidences of chronic valvular disease of the heart. Of these 92 died subsequently, most of them in a recurrent attack of acute infection, giving a mortality of 23 per cent. Of the other cases 29 could not be followed, and only 88 were known to be alive at the end of ten years. The final mortality of rheumatic fever endocarditis, including both the cases which died in the hospital, and those which died subsequently, is represented by the figures, lived 88, died 119, or 52 per cent, and if the cases which also had pericarditis be added, it was 63 per cent.

These figures are given, in order to impress the seriousness of acute endocarditis, due to rheumatic fever in early life.

The prognosis of the form of acute endocarditis which occurs as a complication of some other infection is that of the primary disease. The prognosis of the malignant type of endocarditis is very unfavorable.

TREATMENT.—The general measures of treatment employed in acute endocarditis are those of acute infectious diseases in general, and of rheumatic fever in particular. They include rest in bed, a light diet of liquids and soft solids, and the usual attention to bowels and nursing.

There is very little that we can do which has any direct influence upon the pathological process in the heart. The great essential in treatment is *rest*. Children must be kept in bed, and every form of exertion should be avoided. If there is severe dyspnea or orthopnea they may be propped up with pillows, but should not be allowed to raise themselves. It is often best to feed these patients, rather than to subject them to the slight exertion of feeding themselves. All the measures of sick-room nursing should be carried out without voluntary coöperative movements on the part of the patient. Later in the course of prolonged cases, children may sometimes be allowed to sit up for a time in a chair, provided that they are lifted back and forth from bed to chair, and make no exertion. When a permanently normal temperature, and the disappearance of cardiac symptoms, show that the acute infection has subsided, the very strict rest treatment may be slightly relaxed. The children may then play a little with playthings, and help a little when moved in bed, but the strict rest must be resumed immediately if either fever or any cardiac symptoms reappear. These patients should be kept in bed for a period of from one to three months after the subsiding of all acute symptoms, according to the severity of the case.

An *ice-bag* applied to the precordia is indicated in all cases of acute endocarditis, especially when there is precordial pain, palpitation, or a rapid irritable action of the heart.

The question of whether or not *salicylates* should be given arises in every case. My personal opinion is that the use of salicylates has no influence whatever upon the course of the disease. Furthermore, they may disturb the stomach, which is particularly to be avoided. I do not advocate the use of the salicylates as a routine measure. They are, however, valuable for the relief of pain, and are the most effective weapon against the precordial pain of acute endocarditis. The sodium salicylate should be tried first, in doses proportioned to the age of the child. If it causes nausea or vomiting, aspirin should be substituted. If the salicylates fail to bring satisfactory relief, *codein* should be tried, and if this fails, *morphin* must be resorted to.

Severe cough is best controlled by means of codein or paregoric, morphin being indicated only in the most obstinate cases.

In general, *cardiac stimulants should be sedulously avoided in acute endocarditis.* Digitalis does not "aid in restoring compensation" when given to a patient having an acute infection of the heart. I have often seen its use followed by a markedly increased tendency toward intermitting of the ventricular contractions. We must recognize that in acute endocarditis the symptoms are produced by an acute infection localized in the heart, which runs its appointed course uninfluenced by drugs. We rest a muscle which is the seat of acute inflammation, and do not stimulate it to increased contraction, and the muscle of the heart calls for the same treatment. The mistaken idea that the symptoms of cardiac weakness are due to broken compensation has been the cause of much abuse of digitalis.

But what are we to do when the progress of the case, as shown by increasing signs of cardiac weakness and progressive diminution in the quantity of urine excreted, is steadily becoming more unfavorable? Such conditions are really entirely beyond our aid. The principal indication is to *do no harm*, and the use of digitalis, strophanthus, or caffein may do harm. When the outlook has become really desperate, it is perhaps permissible to try a cardiac stimulant, especially if edema has appeared. *Caffein* is probably the least harmful stimulant in such cases. *Diuretin* may be used without harm when the quantity of urine is small, or when edema is present, but in my experience its effect is very transitory in acute endocarditis. The fact is that the cause of all the unfavorable symptoms lies in a primary weakness of the heart, and the cause of this weakness cannot be reached by medication.

The physician is rarely called upon to treat sudden heart failure in these cases, as when it occurs, it is usually without warning. If there is sufficient time, camphor and nitroglycerin may be tried.

The vomiting, which is so serious a symptom in acute endocarditis, is not to be influenced by medication. The physician should try giving smaller quantities of food at shorter intervals. Sometimes solids are better borne than liquids. In the most severe cases rectal feeding may be necessary.

As the patients are kept in bed until all acute symptoms have subsided, the treatment of convalescence becomes that of chronic endocarditis, and will be described under that disease.

ACUTE PERICARDITIS

Acute infection of the pericardium is comparatively uncommon in infancy and early childhood. In later childhood it is relatively a much more common disease than in adults. Instances of slight pericardial inflammation showing a small amount of fibrinous or sero-fibrinous exudate are a not uncommon finding at autopsy in many acute infectious diseases, without there having been any clinical manifestations during life. There are other more pronounced cases in which a clinical diagnosis is possible, and it is this type which will be considered here.

ETIOLOGY.—The statement is usually made that pericarditis is always a secondary disease. This statement, in my opinion, is based on a fundamental misconception which regards rheumatic fever as primarily a disease of the joints, and pericarditis as a secondary complication. The rheumatic fever of childhood is primarily a disease of the heart just as much as of the joints, and pericarditis, an incident of cardiac infection, should be regarded as a primary manifestation of the disease.

All other forms are indeed secondary processes, complicating various forms of infection. The pathogenic microörganisms reach the pericardium from the primary lesions in two ways. One is by *direct extension* from the adjoining tissues, the lungs, the pleura, or more rarely, the peritoneum. The other way is *through the circulation.*

In the newborn, and in early infancy, pericarditis is most commonly an incident in a general sepsis, the bacteria reaching the pericardium through the blood. The organism may be any of the pus-producing types, but the streptococcus is by far the most common at this age.

In later infancy and early childhood, up to from the fifth to the seventh year, the majority of cases are caused by direct extension from the neighboring lungs, pleura, or occasionally the peritoneum. Most cases are complications of pneumonia or empyema, and the *pneumococcus* is the organism most frequently found, though occasionally streptococci, staphylococci, or influenza bacilli may be present. A certain number of cases occur at all ages as complications of some of the acute specific infections, such as scarlet fever and typhoid fever, without there being evidence of a localized infection in the lungs and pleura. In these cases it is difficult to state whether the infection reaches the pericardium by direct extension or through the blood. Areas of pneumonia and pleurisy are not uncommon complications of the acute specific infections, which areas may be too small to be detected on physical examination, but could still explain infection by direct extension. On the other hand in scarlet fever and typhoid fever, the frequent association of arthritis with pericarditis, and the finding of streptococci or typhoid bacilli in the pericardial exudate, suggest invasion of the pericardium through the blood. Scarlet fever is the most common specific infection in which pericarditis is found as a complication.

In late childhood, after the fifth year, the most common causes of acute pericarditis are *rheumatic fever* and *tuberculosis*. The former disease is the cause of the majority of the cases of pericarditis encountered in childhood. Inflammation of the pericardium is not present in every case of rheumatic fever, nor in anywhere near so large a proportion of cases as is acute endocarditis. It is not often present in the first attack, but the tendency of rheumatic fever toward recurrent attacks, usually involving the heart, accounts for the frequency of pericarditis as a manifestation of this disease. It is to be considered as part of a general carditis, and a primary manifestation of the infection. The pericarditis of tuberculosis occurs through direct extension of the tubercle bacilli from the pleura, or mediastinal lymph nodes, and usually accompanies a tuberculous pleurisy.

Other rare causes of acute pericarditis are trauma, disease of the neighboring bones, and ulceration of a foreign body from the esophagus into the pericardium.

PATHOLOGICAL ANATOMY.—The lesions in the pericardium caused by infection are similar to those of the pleura and serous membranes in general. In streptococcus infections the exudate is usually purulent or seropurulent. In pneumococcus infections the exudate is like that seen in the pleura in lobar pneumonia and empyema. In the milder cases the exudate is fibrinous; in more severe cases there is a thick coating of fibrin on both layers of the pericardium, containing pockets of pus or cloudy serum. In still more severe infections there is, beside the fibrin, a profuse purulent exudate. In tuberculous pericarditis there may be only a deposit of miliary tubercles (this form cannot be recognized clinically), or there may be a serous exudate. The most common form of tuberculous pericarditis is chronic.

In rheumatic pericarditis, the exudate may be only fibrinous (dry pericarditis), or there may be serous exudate in addition (pericarditis with effusion). The exudate is never purulent. The inflammation usually begins near the base of the heart and involves both the visceral and parietal layers of the pericardium. As the pericarditis is part of a general carditis, it is always accompanied by myocarditis.

In all forms of pericarditis, complete recovery usually does not occur. After the infection runs its course, only a part of the exudate is removed by absorption. The remainder undergoes fibrous tissue organization, which varies in extent from slight adhesions to complete obliteration of the pericardial sac. When the results of this process are sufficiently marked to produce secondary changes in the heart or to disturb its function, a disease picture is produced which will be described as chronic pericarditis.

SYMPTOMS.—The diagnosis of acute pericarditis depends mainly upon the results of the physical examination of the chest, the signs depending upon the pathological anatomy rather than upon the etiology. The subjective symptoms, and the more general signs of disturbed cardiac function, have more relation to etiology, and vary according to whether the disease is secondary to some other infection, or is a manifestation of rheumatic fever.

SECONDARY PERICARDITIS.—Acute pericarditis is easily overlooked when it occurs as a complication of some severe infectious disease, such as general sepsis, pneumonia, empyema, or scarlet fever. The symptoms are masked by those of the primary disease. When in the course of such a disease the patient suddenly becomes worse, with precordial pain, restlessness, dyspnea with marked acceleration of the respiration, an anxious expression, a rapid pulse of small volume, marked pallor, and later, cyanosis, pericarditis should be suspected. It must be remembered that in these diseases the patient may die before the characteristic physical signs appear. Also, in pneumonia and empyema the difficulty of diagnosis is even increased by the fact that the physical signs of the disease in the lungs often mask those of the involvement of the pericardium.

PERICARDITIS IN RHEUMATIC FEVER.—When pericarditis occurs as a manifestation of rheumatic fever, the clinical picture is usually much clearer, being that of rheumatic fever in general, unclouded by the symptoms of any other acute infection. The pericarditis in many cases appears as an additional manifestation in a case already recognizable as one of rheumatic fever through the presence of evidences of acute endocarditis or multiple arthritis. In some cases, however, pericarditis is the primary clinical manifestation of the disease. The symptoms are due not only to the inflammation of the pericardium, but also to the accompanying myocarditis, and consequently much resemble those of acute endocarditis. There is at first fever, loss of appetite, restlessness, and precordial pain. The last is particularly severe when the pericardium becomes involved, and may be most distressing. Palpitation, and the signs of " irritable heart " soon develop. In severe cases there is cough, and dyspnea, and eventually there may be enlargement of the liver and edema. In other words, in acute pericarditis all the signs of cardiac insufficiency seen in acute endocarditis are usually present. Precordial pain is apt to be more severe.

When effusion takes place, the precordial pain becomes less, and then often disappears. The formation of a large pericardial effusion is usually stated to cause an increase in the symptoms of cardiac embarrassment, from mechanical pressure. It is true that dyspnea may increase, and the action of the heart may become weaker with a large effusion, but I have more often seen large effusions unaccompanied by signs of increased cardiac weakness. While I cannot deny that a pericardial effusion may become large enough seriously to embarrass the heart, I believe that in most cases signs of increased cardiac weakness are only a coincidence, and are due to the inflammation in the myocardium.

It can easily be seen that a diagnosis of pericarditis cannot be made from the general symptoms. We can only say that there is an acute infection localized in the heart. Recognition of pericardial involvement, and of its extent and character, must depend upon the results of the physical examination of the chest.

PHYSICAL EXAMINATION. FIBRINOUS, PLASTIC, OR "DRY" PERICARDITIS.—The characteristic physical signs are represented by a double to-and-fro murmur, which is not synchronous with systole and diastole, is more superficial than an endocardial murmur, and is often of a rubbing or grating character. This friction-rub is best distinguished from an endocardial murmur from the fact that its rhythm is different from that of the cardial cycle. The impression gained by auscultation is that of two independent mechanical engines in the chest. One, the heart, has a short interval between the first and second sounds, and a longer (diastolic) pause between the second and first sounds. The pericardial friction-rub also has two sounds in each cardiac cycle, but as there is an equal interval between the sounds, and as neither of them is exactly synchronous with either the first or second heart sound, they appear to be produced by a different engine. An endocardial murmur always sounds as if it were a regular part of the cardiac cycle, produced by the heart, and bearing a definite relation to its sounds. Even if one or even two endocardial murmurs are present in addition to a pericardial friction-rub, the latter can usually be distinguished from them by its peculiar rhythm. The sound is localized in a small area, is not transmitted as are endocardial murmurs, and does not replace the heart-sounds. The friction-rub is often intensified by pressure with the stethoscope.

PERICARDITIS WITH EFFUSION.—The principal signs of pericardial effusion in early life are *increase in the area of precordial dulness, diminution in the force of the cardiac impulse and in the intensity of the heart sounds, and an area of dulness and bronchial breathing near the angle of the left scapula.*

The Precordial Dulness.—In considering the area of precordial dulness, it is the absolute or superficial dulness which is of most value in the diagnosis of pericardial effusion in childhood. This superficial dulness is determined by the retraction of the borders of the lungs, which withdraw from the chest walls as the fluid gradually distends the pericardium. The deep dulness is due to the distended pericardium under the lungs, and this to a greater or less degree compresses the lungs, which may be held in position by adhesions. The infant, being less likely to have had previous lesions of the lung and pericardium, gives us the best opportunity for studying the outlines of a pericardial effusion, and the area of superficial dulness is the most valuable physical sign of effusion in infants and in young children.

The earliest sign of pericardial effusion is the appearance of absolute dulness in the fifth interspace to the right of the sternum. The presence of this dulness causes the cardio-hepatic angle to become obtuse instead of acute. Rotch believed that this was not only the earliest, but also the most constant sign of pericardial effusion, but in my experience this is not true. Rotch's investigations were based on experimental injection of the pericardium of the cadaver. In actual pericarditis, the presence of pericardial adhesions may, as shown by autopsy, disturb the distribution of the fluid so that enlargement of the area of dulness to the right may not be present. It usually is present, however, and when present is apt to be the earliest sign to appear.

In moderate effusions there is slight absolute dulness in the fourth right interspace, more marked absolute dulness in the fifth interspace, obtuse cardio-hepatic angle, and enlargement of the precordial dulness to the left. When the cardiac impulse remains visible and palpable, as is usual in moderate effusions, the *dulness in pericardial effusion extends notably beyond the limits of the apex beat.* The larger the effusion, the farther does the area of the precordial dulness extend both to the left and to the right. In small and moderate effusions the area is triangular with a transverse base below.

In very large effusions, the area of precordial dulness is rather circular. The cardio-hepatic angle again becomes acute. The dulness on the right may extend to or even beyond the mammary line.

On the left it may extend to the anterior axillary line, or even to the mid-axillary line. I have seen pericardial effusions so extensive that the dulness on the left merged with the dulness at the base of the left lung behind, filling the entire left chest, pushing up the lung, and giving the same signs as pleural effusion.

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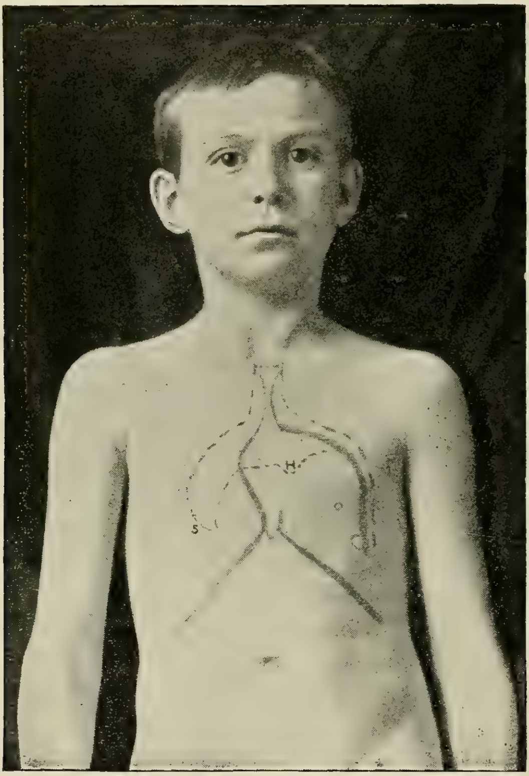


Areas of absolute dulness in enlarged heart, and in distended pericardium.
5, fifth right interspace; H, heart

Cardiac Impulse and Sounds.—With small effusions there is little change in the force or position of the cardiac impulse, or in the intensity of the heart sounds. With larger effusions there is a varying degree of diminution, both in the force of the cardiac impulse, and in the intensity of the sounds. The apex beat may be higher up and farther to the right, even in the fourth space near the sternum. This was formerly attributed to a floating up of the heart, but this explanation is not now accepted, the cause of the phenomenon being that the effusion allows only the front of the ventricle to strike the chest wall. The impulse may disappear to inspection, but still be palpable. In extensive effusions the impulse is usually neither visible nor palpable, and the heart sounds are very feeble. The diminution in the intensity of the heart sounds is not accompanied by a corresponding weakening of the radial pulse,—an important point in diagnosis.

The pericardial friction-rub may still be heard for a considerable time during the formation of the effusion. It is usually present in small effusions, absent in large ones, and may be present or absent in effusions of moderate amount. Obliteration of the intercostal spaces, and even bulging of the precordia, is sometimes seen in the pericardial effusions of children.

Dulness and Bronchial Breathing at the Left Base.—This sign was first described by Ewarts as a characteristic of pericardial effusion. It consists of an area of slight but distinct dulness near the angle of the left scapula, of a size about that of a silver dollar or somewhat larger. Over this area the breathing is distinctly bronchial. Many writers have not considered this a very constant or important sign in pericardial effusion, but in my experience it is very valuable in diagnosis. I have found it invariably present in large effusions, usually present in moderate ones, and often present in small ones. Of course the possibility of consolidation in the lung or of fluid in the pleural cavity must be taken into consideration.

DIAGNOSIS.—In the diagnosis of acute pericarditis the physician is confronted by two problems. One is the recognition of the presence of the anatomical lesion of pericarditis, the other is the recognition of the cause of the disease. The first problem may be called the physical diagnosis, the second the etiological diagnosis, of pericarditis.

PHYSICAL DIAGNOSIS.—The diagnosis of dry pericarditis depends wholly upon the recognition of the pericardial friction-rub. This must be differentiated from endocardial murmurs. The principal points in differentiation are the to-and-fro rhythm of the friction-rub independent of the rhythm of the heart sounds, its grating character, its failure of transmission to the axilla, and the fact that it is often increased by pressure of the stethoscope. In infants and young children, the recognition of the friction-rub is very difficult, and often impossible, on account of the very rapid action of the heart.

The essential point in the diagnosis of pericardial effusion is the presence of an increased area of precordial dulness. The conditions to be considered in differential diagnosis are those which produce increased dulness in the region of the precordia, namely, enlarged heart and lesions in those parts of the lung or pleura which adjoin the precordia.

Enlarged heart gives an increase in the area of the dulness both to the right and to the left. The principal points in the recognition of pericardial effusion are, first, that it usually, though not always, produces absolute dulness in the fifth, and sometimes in the fourth right interspace, a sign almost never seen with enlarged heart. Second, in small and moderate effusions the cardio-hepatic angle is obtuse, while with enlarged heart it is acute. Third, if the cardiac impulse is visible or palpable, the position of the apex beat, or point furthest to the left where the impulse is seen or felt, is displaced outward in enlarged heart; but this is not of much diagnostic value because effusion may be present in addition to cardiac enlargement. The important point is that in enlarged heart the left limit of precordial dulness *corresponds* to the apex beat or extends *very little* beyond, while in *pericardial effusion the dulness usually extends notably beyond the apex beat.* In the absence of a process in the left lung or pleura, this sign is diagnostic. Fourth, diminution in the force of the cardiac impulse and in the intensity of the heart sounds, in proportion to the strength of the radial pulse, points toward effusion. The heart sounds may be fainter than normal, and the impulse weak and diffuse, in a heart enlarged by dilatation, but in children this is not so marked as in adults, and the radial pulse shows some correspondence. In large effusions the heart sounds are often very faint, and the impulse neither visible nor palpable; this is almost never seen in dilatation of the heart in childhood. Fifth, the area of dulness and bronchial breathing in the left back often found in effusion, is almost never caused by enlarged heart alone. Sixth, the presence of a friction-rub in doubtful cases points toward effusion, but confirmatory signs are needed, because dry pericarditis is often seen with enlarged heart. Seventh, in very large effusions the area of precordial dulness is so greatly increased, that, although the cardio-hepatic angle is acute, the condition is not likely to be taken for enlarged heart. In such cases the cardiac impulse usually cannot be detected, the heart sounds are very faint, and the area in the left back is very distinct.

Pleuritic effusion is, I think, in childhood more often confounded with pericardial effusion than is enlarged heart. With free fluid in the pleural cavity, the line of dulness passes entirely around the chest, while with all but the largest pericardial effusions, there is resonance beyond the area of precordial dulness. I have seen extremely large pericardial effusions in which the precordial dulness on the left merged with the dulness in the left back, mistaken for pleurisy. In such cases the extension of dulness to the right is mistakenly attributed to pushing over of the heart. The determining points in the correct diagnosis of pericardial effusion in such cases are the absence of cardiac impulse, the faint heart sounds, and the fact that the point where the heart sounds are heard best is not displaced toward the right. When a pleuritic effusion is encapsulated adjoining the precordia, the physical diagnosis is more difficult, as the fluid in the pleura gives dulness on one side of the precordia with resonance beyond, while the displacement of the heart gives dulness on the other side. In such cases, displacement of the impulse and point of greatest intensity of the heart sounds points toward pleurisy, while feeble impulse, faint heart sounds, and a possible friction-rub point toward pericarditis. Roentgenograms are a great aid in the diagnosis of these difficult cases. When pericardial effusion occurs with pleuritic effusion, as often happens in empyema, the recognition of the former is still more difficult, unless a typical pericardial friction-rub be present. The important points in pericardial effusion are absolute dulness in the fifth right interspace with an obtuse cardiohepatic angle when the empyema is on the left, extension of the dulness to the left notably beyond the apex beat when the empyema is on the right, and weak impulse with faint heart sounds. Of course a typical pericardial friction-rub is the greatest aid in the recognition of these cases, but this is often absent, and pericardial effusion complicating empyema is frequently overlooked.

Pneumonia without pleuritic effusion presents less difficulty in differential diagnosis. When pericardial effusion is absent, the precordial dulness is not increased on the other side, unless endocarditis and enlarged heart are present. The bronchial breathing and râles often permit the recognition of the consolidation. The other signs of pericardial effusion are absent, unless it be present as a complication. Roentgenograms give a valuable confirmation of the diagnosis.

The presence of pericardial effusion can be positively confirmed by aspiration of the pericardial sac. This procedure is not warranted in all cases merely to establish the physical diagnosis, and its indications will be considered under the etiological diagnosis of the disease.

ETIOLOGICAL DIAGNOSIS.—When the physical examination reveals the presence of fluid in the pericardial sac, the prognosis and treatment of the case depend largely upon its cause. It is essential to determine if the fluid be serous or purulent. In rheumatic fever the fluid is always serous. The diagnosis is based on the considerations which determine that of rheumatic fever in general, the principal point being the presence of an acute infectious disease in later childhood, with symptoms localized in the heart, in the joints, or in both, and without evidences of pneumonia, or of any other specific infection. Diagnostic aspiration is not justified in cases which are thus diagnosed.

The etiological diagnosis of secondary forms of pericarditis depends upon the nature of the primary infection. When the pericarditis is a complication of general sepsis in infancy, or of empyema, the exudate is usually purulent, and exploratory aspiration is positively indicated. When the pericarditis complicates pneumonia, or an acute specific infection, the exudate may be serous at first, but later becomes purulent. It is so important in these cases to determine whether pus is present, that exploratory aspiration should always be performed. The diagnosis of tuberculous pericarditis depends on the recognition of the primary disease.

The technic of exploratory puncture of serous cavities has been described in Division II. Opinions have differed widely as to the best point for entering the pericardial sac. In the order of the writer's preference, the available sites for the puncture are: (1) In the fourth or fifth left intercostal space near the sternum; (2) in the same spaces outside the left nipple, but within the line of precordial dulness; (3) high in the angle between the xyphoid cartilage and the left costal border, the needle being directed backward until it has penetrated as far as the posterior surface of the costal cartilage, and then turned sharply upward; (4) in the fifth right intercostal space about an inch from the sternum.

After exploratory puncture, any fluid should be carefully examined as to its bacteriology and cellular constituents.

PROGNOSIS.—Purulent pericarditis in children, whether due to the pneumococcus, streptococcus, or some other organism, is usually fatal. It is practically always a fatal complication in infants.

In pericarditis due to rheumatic fever, the prognosis is that of rheumatic fever in general. The outlook as to life in any one attack is fairly good. Nevertheless it must be remembered that pericarditis is a serious manifestation of the disease. The fact that the inflammation involves the pericardium usually means that there is a severe involvment of the myocardium, and it is because of the myocarditis that children so often die in acute rheumatic fever. The immediate mortality in 58 cases of rheumatic fever with pericarditis was 31 per cent, as against 12 per cent in the cases having acute endocarditis only. The subsequent mortality in the cases which recover from the acute attack is that of any child who has had an attack of rheumatic fever. A certain number of cases die from a recurrent attack of the disease. In the cases which escape, a certain amount of pericardial adhesion is left behind. In many cases this is not sufficient to embarrass the heart. In some cases the clinical picture of chronic adhesive pericarditis develops, with its very uncertain outlook.

TREATMENT.—In the pericarditis of rheumatic fever, the treatment is precisely that described for acute endocarditis with severe symptoms. The ice-bag should be kept constantly applied to the precordia, unless the children will not tolerate it, when dry heat should be substituted. The salicylates are usually insufficient to control pain, and opium has to be used. It is also useful in quieting the cough. Digitalis is contraindicated as in acute endocarditis.

Alcohol is contraindicated. Caffein may be tried in threatening cardiac failure.

The only question which arises in acute pericarditis is whether the effusion should be relieved by aspiration. In the majority of cases the fluid is absorbed spontaneously, even though its quantity be large. It is doubtful whether even a large effusion often causes a serious mechanical embarrassment of the heart. The symptoms of cardiac weakness are probably usually due to the acute myocarditis rather than to the pericardial effusion, even when the quantity of fluid is increasing. Even progressive cardiac failure is usually due to the infection. Nevertheless, in a case with a large effusion running an unfavorable course, with increasing signs of cardiac weakness, aspiration of part of the pericardial effusion is indicated. If very rapid breathing or cyanosis is present, aspiration should always be performed. If part of the fluid is removed, the rest will usually be readily absorbed. The guide as to aspiration is the severity of the circulatory disturbance, not the size of the effusion as indicated by physical examination.

The only form of treatment holding out any hope of cure in *purulent* pericarditis, is surgical incision and drainage.

CHRONIC ENDOCARDITIS

(Chronic Valvular Disease of the Heart)

ETIOLOGY.—The cause of chronic endocarditis in early life is almost invariably a preceding acute endocarditis. Common causes of chronic endocarditis in later life, such as syphilis, alcohol, arteriosclerosis, and so forth, are not seen in childhood. As the most common cause of acute endocarditis in childhood is rheumatic fever, most cases of chronic valvular disease are to be traced to this infection. It follows from this that chronic endocarditis is seen most often in the latter part of childhood.

PATHOLOGICAL ANATOMY.—Although the morbid process may attack the parietal endocardium, it is the valvular lesion that is of such clinical importance as to become the prominent feature in chronic endocarditis. The process most frequently affects the valves on the left side of the heart, the mitral valve being the one usually involved in childhood. It is a slow, insidious process, manifested by thickening, induration, and adhesions, followed by a contraction of the valvular segments. Later, lime-salts may be deposited in the valves and this results in rigidity. As the sclerotic changes increase, the deformity of the segments becomes greater and results either in imperfect closure of the valves (valvular insufficiency), allowing a regurgitation of the blood, or the orifice is narrowed (valvular stenosis), causing obstruction to the blood-current. We meet, especially in children, with lesions at the mitral orifice in which the valves are curled and thickened so that they are insufficient, although there may be no narrowing of the orifice. The chordae tendineae gradually become shortened. The apices of the papillary muscles are enlarged, the endocardium becomes thickened, and marked changes following the valvular lesions take place in the walls of the heart.

The frequency of the various valvular lesions is represented by the following table showing the lesions in a series of 262 cases.

TABLE 64

Occurrence of the Various Valvular Lesions

	CASES
Mitral insufficiency alone	165
Mitral stenosis alone	5
Aortic insufficiency alone	1
Mitral insufficiency and stenosis	79
Mitral and aortic insufficiency	8
Mitral insufficiency and stenosis with aortic insufficiency	4

DILATATION, HYPERTROPHY, AND COMPENSATION.—The preceding acute infection involves the myocardium as well as the endocardium, and the weakening of the muscular walls caused by the myocardial infection produces an enlargement of the cavities of the heart, which is called *dilatation*. After recovery from the acute infection, the chronic valvular lesion which persists presents a mechanical embarrassment to the circulation, which causes the dilatation to persist. If there were no change in the muscular walls of the heart, the valvular lesion plus the dilated cardiac chambers would continue to produce symptoms of cardiac weakness. The heart muscle, however, has the power of *hypertrophy*, as a result of which the muscular walls of the heart increase to a thickness above the normal. This muscular hypertrophy is of course accompanied by an increase in the mechanical power of the heart, and is Nature's method of attempting to nullify the effects of a permanent mechanical lesion. When the hypertrophy of the myocardium is sufficient, the circulation can be carried on normally, in spite of the mechanical obstruction caused by the valvular lesion. In such a case the valvular lesion is said to be "*compensated*," and compensation is complete when the circulation is wholly normal. When there are persistent symptoms of cardiac weakness, and of the resulting disturbance of circulation, compensation is partial or incomplete. When, after a period of good compensation in a case of chronic endocarditis, symptoms of cardiac weakness and circulatory disturbance appear, the condition is spoken of as "*broken compensation*" or "*failure of compensation*."

In children, the power of the heart to establish effective compensation through myocardial hypertrophy is exceptionally good. The contrary has often been stated in medical literature. The liability of children to attacks of circulatory failure from cardiac weakness has led to the impression that compensation is not good in childhood. These attacks, however, as we have seen when considering acute endocarditis, are due to recurrences of an acute infection to which children are peculiarly liable, and they cannot be brought forward as evidence that the process of hypertrophy which leads to compensation is not complete. The statement has also been made that the demands upon a child's heart through the necessities of growth and development are such that they interfere with the establishment of compensation. This also is not true. The very fact that the child's heart has a capacity for growth, means that it has also an increased capacity for compensation in the face of a mechanical lesion. The process of hypertrophy through which compensation occurs is closely associated with that of normal development. It is a rule of early life that Nature's power of neutralizing and off-setting the effects of a chronic lesion are greatest during the period of development. After the period of growth is over, compensation by means of processes of growth takes place less readily. Not only is cardiac compensation when gained in childhood more complete, but it is the better the earlier in life that the valvular lesion is acquired. When children acquire chronic endocarditis in early childhood, there is still left before the child a considerable part of the period of growth, during which a very complete compensation, much more complete than could be gained later, may be attained, providing that no disturbing factors intervene. In a case of chronic endocarditis acquired in early childhood, the subsequent period of development permits a mutual adaptation to take place between the growing child and the damaged but growing heart. The child develops to fit its heart, while the heart develops to fit the child, in spite of the valvular lesion.

What are the disturbing factors which prevent the establishment of satisfactory compensation? There are three: 1. Recurrent acute infection; 2. poor nutrition; 3. overexertion. These three are given in the order of their importance. The liability of children to recurrent attacks of rheumatic fever with cardiac involvement has been mentioned repeatedly. The acute infection in these cases does not leave behind a permanent lesion of the myocardium, nor permanently damage its capacity for hypertrophy. It does, however, lead to a fresh dilatation of the heart, and the work done by Nature in her effort to establish compensation, is all undone, and has to be performed all over again. This happens with each acute attack. Meantime the precious period of growth is fast passing, and little of it may remain when the last attempt at the establishment of compensation is made. The nutritional factor is next in importance. If the nutrition of the growing child is poor, normal development suffers.

The nutrition of the heart is affected with that of the whole body, and the developmental processes in the heart which cause compensatory hypertrophy are interfered with. In such cases we must trace the failure of establishment of satisfactory compensation back to the manifold causes of impaired nutrition, among which are numbered hygienic and dietary factors, as well as the various diseases which affect nutrition. The third factor is that of overexertion, in which, before compensation is adequate, too great a load is thrown upon the heart. This may be the cause of failure of compensation in childhood, and is important, but its frequency and importance have been somewhat overemphasized in proportion to the first two factors.

What are the causes of broken compensation, that is, of the development of cardiac insufficiency in a case of chronic endocarditis in which compensation has previously been good? The most common cause of cardiac insufficiency in such cases is a new attack of rheumatic fever. I think, however, that it is better not to apply the term broken compensation to cases in which the cardiac failure is due to acute cardiac infection. The term broken compensation has been so much associated with overexertion as a cause, that it has come to imply a mechanical cause for the disturbance. Furthermore, in acute endocarditis the same manifestations of cardiac weakness occur in cases in which there is no previously acquired chronic endocarditis. The symptoms are due to the acute endocarditis, whether or not there has been a previous chronic endocarditis.

Failure of compensation in chronic endocarditis is produced by a disturbance in the balance between the power of the heart and the work which it is required to perform. *Overexertion* is the most common cause. This causes dilatation, and the resulting cardiac weakness can only be compensated by increased hypertrophy, which may not be possible. Compensation may fail in children in whom there is no change in the amount of physical exertion taken. In such cases we must look to causes which affect the muscular power of the heart. In the various acute illnesses due to the specific infections, the heart muscle is affected by the bacterial toxins circulating in the blood. These may produce myocardial degeneration and dilatation even when no chronic valvular lesion is present, and when chronic endocarditis is present, much more serious disturbance of cardiac function may be produced. Still, many children with chronic valvular disease of the heart may undergo an acute illness without a breakdown of compensation. Beside the acute diseases, the most common causes of failure of compensation are to be found among the many factors which affect nutrition and development.

SYMPTOMS.—The symptoms of chronic valvular disease of the heart depend upon the degree of compensation which is present. For convenience of clinical description, and of indicating treatment, cases of chronic endocarditis may be divided into three groups: 1. Cases in which compensation is complete; 2. cases in which compensation is partial or incomplete; 3. cases in which compensation has failed.

It must be remembered, however, that these divisions are not bounded by hard and fast lines, but run into one another, so that every degree of compensation is met with from the most complete to that of total failure.

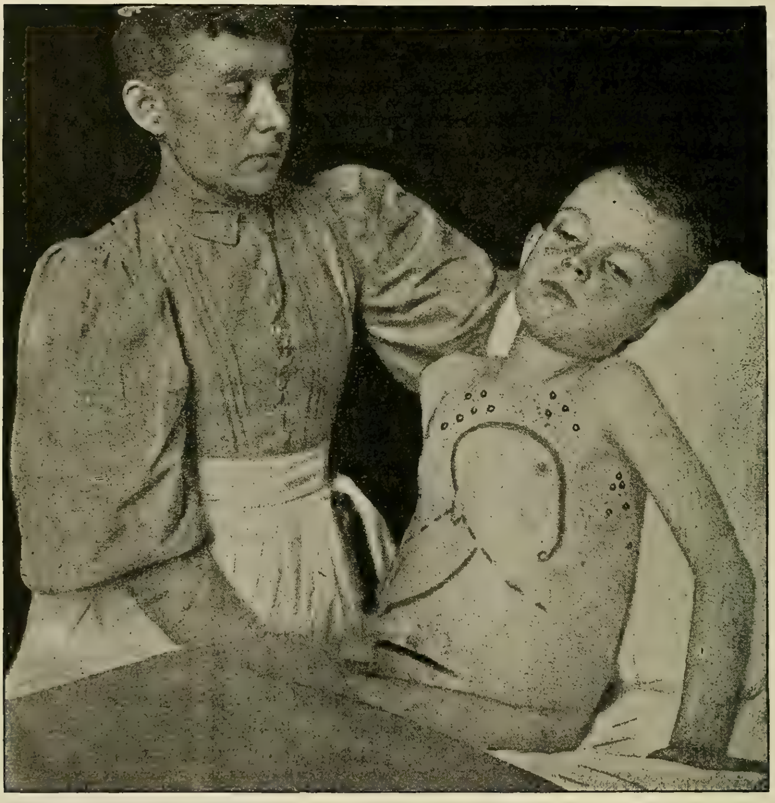
COMPLETE COMPENSATION.—In this stage there are no notable symptoms. The disease may exist for months or years without being recognized. In many such cases the diagnosis is made as a result of some routine physical examination, undertaken at school, or because the child wants to engage in some form of competitive athletics, or because of the existence of some acute illness. In other cases the child may be brought to the physician for examination because of various vague symptoms, the parents thinking that the child is becoming " run down," without being able to give any very definite reasons for their belief. In some cases there is a definite history of shortness of breath on violent exertion, but more often not, the only complaint being that the child gets tired easily, and seems unduly exhausted after exercise. The essential feature, however, of the stage of complete compensation in a child, is that none of the symptoms have been severe enough to interfere with the normal activities of a child of that age.

PARTIAL COMPENSATION.—In this stage there are symptoms marked enough to attract the attention of the parents, and perhaps to limit the activities of the child's daily life. Shortness of breath on exertion is the most notable symptom. At times there may be other symptoms, such as precordial pain, attacks of palpitation, cough, headache, epistaxis, and loss of weight. Anemia is often prominent. Often in children there is seen after exertion an attack of faintness, or of complete exhaustion.

In this stage the symptoms are very variable. There are periods in which they are quite marked, approaching those of failure of compensation; at other times the condition of the child approaches that of the stage of complete compensation. The course of the disease varies according to the cause which prevents the establishment of complete compensation. It is usually stated that children in this condition tend to get worse rather than better. This is because the cause, whether it be continual overexertion, or the hygienic factors which prevent proper nutrition, is not removed.

FAILURE OF COMPENSATION.—When this occurs the symptoms of cardiac insufficiency are more severe. There is marked dyspnea or orthopnea, and cough. Often there is profuse frothy expectoration, which may be bloody. The further backing up of the blood leads to congestion of the systemic venous system, which manifests itself in *edema.* The edema usually begins in the feet and extends to the ankles and legs, but may begin in the face. In severe cases there may be general anasarca. Fluid may accumulate as a transudate in the serous cavities, pleural or peritoneal, or both. There is usually enlargement and tenderness of the liver, and there may be enlargement of the spleen. The congestion of the veins of the gastro-intestinal tract may cause symptoms of indigestion, and vomiting is a serious symptom in severe cases. The urine is scanty, and contains albumin and other evidences of passive congestion of the kidneys. There may be visible congestion of the superficial veins,

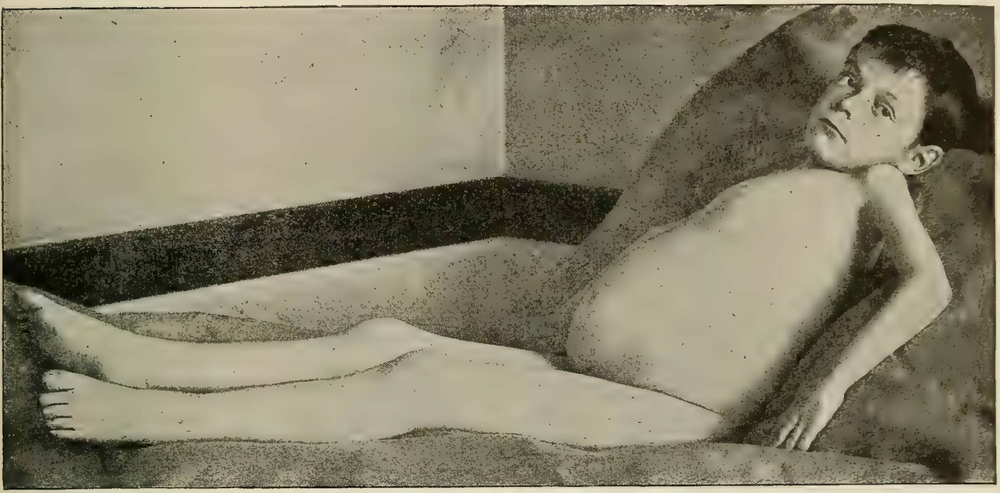
FIG. 222



Chronic recurrent endocarditis. Mitral insufficiency. Disturbance of compensation. Dilated heart. Enlarged liver. Edema of lungs. Ascites. Male, 10 years old and cyanosis, although the latter is rare. There may be headache, attacks of dizziness, or fainting spells.

Failure of compensation may occur fairly suddenly as a result of overexertion in any child with a chronic valvular lesion. Progressive failure is most apt to occur in children at about the age of puberty, possibly because of the increased demand made upon the heart at that age. The symptoms may increase in severity until death occurs, or at any time may become less severe, with eventual reestablishment of compensation. The cause of death is most often sudden failure of the heart, probably due to an acute dilatation. It may be due to an intercurrent disease, to embolism, or to edema of the lungs.

FIG. 223



Chronic endocarditis. Mitral insufficiency. General edema and ascites. (Before treatment)

PHYSICAL SIGNS.—There are two important physical signs which are found in all stages of chronic endocarditis. These are, (1) one or more *endocardial murmurs*, and (2) signs of *enlargement of the heart.*

MURMURS.—The endocardial murmurs of children do not differ in any important particular from those of adults. For this reason, their description is, perhaps, superfluous. Nevertheless, for purposes of completeness, a brief description will be given of the most important characteristics of the murmurs produced by the several valvular lesions.

The murmur of *mitral insufficiency* is by far the one most common in childhood. This murmur is systolic in time, and is either synchronous with the first heart sound, or immediately follows it. It may wholly or in part replace the first heart sound. It is loudest at the apex, is heard all over the precordia, is transmitted to the left into the axilla, and is usually audible at the angle of the left scapula. It may be loud enough to be heard all over the chest. Its intensity varies. Unless there is a preexisting chronic lesion, the murmur of acute endocarditis is comparatively faint. As the condition of chronic endocarditis supervenes, and as compensation becomes established, the murmur grows progressively louder, and then remains unchanged. When compensation partially or completely fails, the murmur may grow louder or fainter. The murmur is usually of a "blowing" character, but may be "musical," although musical murmurs are less common in children than in adults.

The murmur of *mitral stenosis* is much less common than that of mitral insufficiency, and when present, is usually associated with the latter, though it may exist alone. It is presystolic in time, beginning during the diastolic pause, and terminating abruptly with a sharply accentuated first heart sound. Its quality is wholly different from that of the murmurs of mitral and aortic insufficiency, being prolonged, rough, and rolling. It is loudest at or near the apex, and it is not transmitted into the axilla, but is heard only over a circumscribed area. It causes a distinctly palpable purring presystolic thrill, which terminates sharply with the impulse. The presystolic murmur is loudest in cases with well marked cardiac hypertrophy. In dilatation, whether caused by a fresh acute endocarditis, or by failure of compensation, it becomes fainter, and it may disappear entirely; but the diagnosis of mitral stenosis can still be made from the intense accentuation of the first heart sound at the apex, which is characteristic of this lesion, and by a reduplication of the second sound at the apex, which is very common.

In some cases of mitral stenosis of milder grade, there is no typical presystolic murmur, but there is a short blowing murmur which accompanies or immediately follows the second heart sound. This is heard most often after acute endocarditis, in the stage before the development of hypertrophy brings out the typical presystolic murmur, and is often mistaken for that of aortic insufficiency. It is, however, heard only over a limited area near the apex, is shorter, is not transmitted into the carotids, and does not replace the aortic second sound.

The murmur of *aortic insufficiency* is much less common in children than in adults. Aortic insufficiency is rarely the sole lesion present, and aortic murmurs are usually present in addition to mitral murmurs. The murmur is diastolic in time, blowing in quality, and prolonged, beginning with, or immediately after, the second sound, and often extending throughout the diastolic pause. It entirely replaces the aortic second sound, and may be so loud that it conceals the pulmonic second sound. It is usually loudest in the third space at the left border of the sternum, but is heard all over the precordia, and may often be heard in the axilla. It is also heard in the carotid arteries. Other signs seen with aortic insufficiency in children as in adults, are intense throbbing of the carotids, the "Corrigan's" or "water-hammer" pulse, the capillary pulse, and the "pistol-shot" sound in the femoral arteries.

Aortic stenosis is very rare in early life, and never occurs as the only lesion. The murmur is systolic, loudest at the second left interspace, transmitted into the carotids, and accompanied by a palpable systolic thrill.

Tricuspid insufficiency does not represent a primary valvular lesion in chronic endocarditis, but is relative, due to dilatation of the right ventricle, secondary to the disease in the left side of the heart. A relative tricuspid insufficiency may also occur in certain diseases of the lungs, such as empyema, chronic interstitial pneumonia, or chronic pleurisy. The murmur is systolic, loudest over the lower part of the sternum, and usually heard only over a small area. The jugular veins are prominent, and may show systolic pulsation. There may be a palpable systolic pulsation over the liver.

No other valvular lesions are seen in chronic endocarditis.

CARDIAC ENLARGEMENT.—The area of precordial dulness is usually enlarged in chronic endocarditis, no matter what the particular valvular lesion may be. Mitral lesions are compensated mainly by hypertrophy of the right ventricle, aortic lesions by hypertrophy of the left ventricle. In mitral insufficiency there is usually some enlargement of the left ventricle as well as of the right. Theoretically, with hypertrophy of the right ventricle the precordial dulness should be enlarged to the right, while with hypertrophy of the left ventricle it should be enlarged chiefly to the left. In children, however, this distinction is not so marked as in adults, and the precordial dulness is enlarged both to the right and to the left with both mitral and aortic lesions. The impulse and apex beat are found further to the left than the normal position, and the dulness to the left extends to a varying distance beyond the normal limit. On the right, the absolute or superficial dulness extends to the right sternal border or even beyond in the third and fourth spaces, and the relative or deep dulness is displaced to the right for a varying distance, even to the right nipple in some cases.

OTHER SIGNS.—The most important confirmatory sign with mitral lesions is accentuation of the pulmonic second sound. This is almost invariably present. The impulse may be heaving and forcible, particularly in cases with only partial compensation, or in cases with fully compensated aortic lesions. When aortic insufficiency is present, the enlargement of the precordial dulness to the left, and the displacement to the left and downward of the apex beat, are very marked. With fully compensated mitral disease, the impulse is often not notably forcible, and the enlargement of the precordial dulness not marked. In failure of compensation, the impulse is often diffuse and undulatory, and may be feeble, and the second heart sounds are often reduplicated at the apex.

The radial pulse is not a very good indication of the functional power of the heart in the chronic endocarditis of early life. In cases with full or partial compensation, both the volume and tension of the radial pulse are good. In failure of compensation the volume of the pulse may remain good, and the tension may be either high or low.

Other signs, indicative of partial or complete failure of compensation, for which the physician should look in the physical examination of a case of chronic endocarditis are the following: Cough with frothy sputum; enlargement and tenderness of the liver; dyspnea or orthopnea; edema of the extremities or face; ascites; signs of fluid in the pleural cavities; diminution in the quantity of urine.

DIAGNOSIS. IN THE STAGE OF COMPENSATION.—In these cases the patients do not present symptoms of illness, and the problem of diagnosis is the interpretation of the cardiac murmur found by the physician. If the murmur is of a typical presystolic or diastolic type, there is no difficulty in recognizing mitral stenosis or aortic insufficiency. If there are more than one murmur, the diagnosis of chronic valvular disease is clear, and it is only necessary to deduce the particular lesions from the characteristics of the murmurs. It is in cases having a systolic murmur only that there is difficulty. The physician must distinguish the murmur of mitral insufficiency from the *accidental murmurs* on the one hand, and from the murmur of *congenital cardiac disease* on the other.

The chief points in the differentiation of mitral systolic murmurs from accidental murmurs, are the presence with the former of an increased area of cardiac dulness, of transmission of the murmur into the axilla, and of accentuation of the pulmonic second sound. Roentgenograms will often confirm the presence of cardiac enlargement when the results of percussion are doubtful. The history of a preceding acute attack with either cardiac, articular, or choreic symptoms is strongly confirmatory of acquired valvular disease.

The only congenital cardiac anomaly likely to be mistaken for acquired mitral insufficiency, is defective interventricular septum. This lesion is characterized by a systolic murmur and cardiac enlargement. In infants, most cases with this symptom complex have a congenital lesion. In older children, most cases represent chronic endocarditis with mitral insufficiency. The murmur may be harsher in congenital disease. A history of a preceding rheumatic fever attack, or of scarlet fever or some definite specific infection, favors mitral disease.

IN COMPLETE OR PARTIAL FAILURE OF COMPENSATION.—In these cases the patient presents the easily recognizable symptoms of cardiac insufficiency, and the physical examination at once reveals the presence of a valvular lesion. The only point the determination of which is important in prognosis and treatment, is whether the symptoms and signs are due to an acute or to a chronic endocarditis. Three conditions are possible. (1) An attack of acute endocarditis, that is, of rheumatic fever localized in the heart, without a preceding chronic endocarditis. (2) Chronic endocarditis with a fresh acute infection. (3) Chronic endocarditis with failure of compensation due to relative overload. The first two conditions, from the point of view of prognosis and treatment, are classified as acute endocarditis. They are recognized mainly by the presence of a febrile reaction such as is seen in acute infections. The coincident presence of evidences of arthritis, confirms the diagnosis. These cases have been described under acute endocarditis.

When the cardiac symptoms are unaccompanied by evidences of an acute infection, we are justified, when the physical signs of a valvular lesion are present, in making a diagnosis of chronic endocarditis with disturbance of compensation from relative overload.

PROGNOSIS.—In chronic endocarditis the diagnosis is by no means so troublesome a problem as is the prognosis and treatment.

In the first place, a distinction must be drawn between cases in which the origin of the cardiac disease was an attack of rheumatic fever, and those in which the original acute endocarditis occurred as a complication of scarlet fever or some other acute infection. In the latter class of cases, which form a small minority in the chronic endocarditis of early life, the prognosis is much better than in the cases of rheumatic fever origin. The chief factor which plays a part is the ability of the growing child to establish effective compensation for a chronic mechanical lesion of the heart. This ability is the greater the younger in life that the lesion is acquired. Provided that the subsequent nutrition of the child is not disturbed, and that the amount of physical exertion allowed is properly regulated, there is nothing to disturb the progressive establishment of a high degree of compensation. Occasionally compensation fails, through the occurrence of intercurrent disease, of imprudence as to exertion, or of chronic nutritional disturbance. Even in these cases the outlook is fairly good as to recovery with proper treatment. In the majority of cases, often without proper treatment, compensation does not fail, and the patients enter adult life well equipped as to the power of the heart to meet adequately the strain of a fairly active existence.

The problem of prognosis is entirely different when chronic endocarditis is acquired as a result of rheumatic fever. There are two conflicting factors which influence the prognosis. The first is the power of children with chronic valvular disease of the heart to attain a very effective compensation, which power is the better the earlier in life that the lesion is acquired. The other is the liability of children to recurrent attacks of rheumatic fever with acute endocarditis, which liability is the greater the earlier in life that the first valvular lesion is acquired. This means that a *distinction must be drawn between the prognosis as to life during childhood, and the prognosis as to both life and efficiency after adult life is reached.*

During the remainder of childhood, that is, up to the age of about fourteen years, the child with chronic endocarditis is menaced by two possibilities. One is the constant liability to a recurrent attack of rheumatic fever with a fresh acute endocarditis. The other is the liability to failure of compensation from relative overexertion, chronic disturbance of nutrition, or acute intercurrent disease. Of these two, the first menace is by far the more serious and important. The majority of children with acute endocarditis who die, die during childhood, and die from an attack of acute endocarditis. In a series of 55 consecutive fatal cases of endocarditis studied at the Children's Hospital, 45 had fever and symptoms of rheumatic fever at the time of death, while only 10 died of failure of compensation from other causes. Out of a series of 264 consecutive cases of rheumatic fever, 17 died in the first attack, 47 recovered without subsequent recurrences, and 200 had subsequent attacks. It follows from these facts, that the prognosis during childhood of chronic endocarditis of rheumatic fever origin is that of rheumatic fever in general. Out of 235 cases followed for ten years, the final outcome was, lived 88, died 147. Of the 147 fatal cases, 140 died before the age of fourteen years, and only 7 died after that age.

The significance of these facts may be summarized as follows: In children with chronic endocarditis acquired as a result of rheumatic fever in childhood, the chances are more against than in favor of the eventual survival of the child. The chances are in favor of the unfavorable ending coming before the expiration of childhood, the danger being partly from relative overload before the establishment of effective compensation, but chiefly from the liability to recurrent attacks of acute endocarditis.

The prognosis as to life in a child who has escaped the dangers which menace him during childhood, and who enters adult life with a heart permanently damaged by chronic endocarditis, is entirely different. He is not nearly so liable to recurrent attacks of rheumatic fever, and if such attacks do occur, the infection is much less likely to be localized in the heart. The prognosis depends upon the degree of compensation which has been attained in childhood. This in turn depends upon the length of the period between the last attack of rheumatic fever and puberty. The longer this period, the better is the compensation. In my series, 95 cases entered adult life with chronic endocarditis. Of these 88 were alive at the end of ten years.

The disability following rheumatic cardiac disease acquired in childhood is a question of the greatest importance. The survival of 88 patients observed for ten years, all of whom had entered young adult life, gives a basis for conclusion. The amount of disability is shown in Table 65.

TABLE 65

Disability in Cardiac Disease Acquired in Childhood Observed in Young Adult Life

	CASES
Disability great	2
Disability slight	9
Disability none	77

The patients with great disability were unable to work or to lead normal lives. Both of them had been in an adult hospital with broken cardiac compensation. Cardiac symptoms are brought on by comparatively slight exertion. The patients with slight disability had dyspnea on exertion, and some had occasional cough and slight edema. None of them had had an attack of severe broken compensation. They were able to work at sedentary occupations. 77 patients had no disability. Most of them were working, and all of them were apparently leading perfectly normal lives, having had no cardiac symptoms since their childhood. My last set of reports contained many references to activity in dancing on the part of girls, and in basketball and baseball on the part of the boys. One of the latter asked my permission to enter the 25-mile Marathon race. All of these 88 patients still had their cardiac murmurs when I last saw them.

It seems to me that the general amount of disability seen in rheumatic cardiac disease acquired in childhood is remarkably small. I believe it to be very much less than the disability following rheumatic endocarditis acquired in adult life. In adult out-patient departments and in general hospitals, the majority of patients suffering from broken compensation or serious disability from chronic endocarditis, can trace the origin of their lesion, if at all, to some attack of rheumatic fever occurring since they had entered adult life. On the other hand, in every adult out-patient department are seen many patients who are seeking medical aid on account of some affection other than cardiac, in whom, in the course of routine examination, some valvular lesion of the heart is found. In a large number of cases these patients declare that they have never suffered from cardiac symptoms, and have no recollection of any attack of rheumatic fever. I believe that in very many of such patients, the cardiac lesion represents the sequela of an attack of rheumatic fever which occurred in childhood, at an age when the arthritic manifestations are often so much less pronounced, that they are not remembered. These patients, leading normal lives, yet with every evidence of organic cardiac disease, are common. They correspond to the 77 cases in my series in which no cardiac disability persisted.

The significance of these facts may be summarized as follows: The prognosis both as to life and as to the ability to lead active useful lives is relatively good in children who enter adult life with chronic endocarditis acquired in childhood.

The above considerations as to prognosis have to do with the general future outlook in the disease, and apply to the cases encountered in the stage of compensation, or who have recovered from acute endocarditis, or from failure of compensation. We must now consider the prognosis in a particular attack of partial or complete failure of compensation. It depends mainly upon whether the possibility that the symptoms of cardiac insufficiency are due to an acute infection of the heart can be excluded with certainty. If this possibility cannot be excluded, the prognosis is that of acute endocarditis, which has already been discussed. If the factor of fresh acute infection can be excluded, the prognosis as to improvement and restoration of compensation is much better, provided that the proper treatment be employed. The outlook depends somewhat upon the cause of the failure of compensation. If the cause be overexertion without an underlying nutritional factor, the case is likely to run a more favorable course under proper treatment.

The character of the valvular lesion has some influence on prognosis. When failure of compensation occurs in a child with an aortic lesion, the course of the disease is apt to be less favorable than when the mitral valve only is affected.

The severity of the symptoms of failure of compensation has some bearing on the outlook in an individual case. The best measure of the prognosis of these cases is the daily quantity of urine excreted. Progressive diminution in the quantity of urine under proper treatment is always an unfavorable sign, while a steadily maintained large or increasing urinary excretion is correspondingly favorable. It must be remembered, however, that an increase in the quantity of urine, following the giving of a diuretic or the changing of the diuretic, is not necessarily a sign of permanent improvement, as such diuresis is often only temporary. There must be a permanent increase in the excretion of urine, before a favorable conclusion can be drawn with certainty.

The severity of the dyspnea is not of so much prognostic significance as is the severity of the signs of passive congestion. The amount and persistence of edema under treatment are important prognostic indications. Vomiting is always a bad sign in cardiac insufficiency in childhood, and the onset of uncontrollable vomiting is often the most conspicuous of the events which foreshadow a fatal ending.

In general, the physician must give a guarded prognosis in a case suffering from failure of compensation. There are too many possible etiological factors which are beyond the reach of his knowledge. Still the majority of these cases, under proper treatment, recover from the immediate attack.

TREATMENT.—The treatment of chronic valvular disease of the heart varies with the different stages in which the disease is encountered. It is convenient to consider the indications for treatment in the following stages: (1) After convalescence from acute endocarditis; (2) in the stage of complete compensation; (3) in the stage of partial or incomplete compensation; (4) in the stage of failure of compensation.

CONVALESCENCE FROM ACUTE ENDOCARDITIS.—Patients with acute endocarditis are kept in bed for a period of from one to three months after a permanent return of normal temperature and the disappearance of all signs of cardiac insufficiency. During this period the aim of treatment is to establish the highest degree of effective compensation which can be attained. The prolonged rest guards the heart from the disturbance caused by relative overload, and this is essential in the establishment of effective compensation. On the other hand, the nutrition of the heart is important, and depends upon general normal development and nutrition. Rest in bed is not the condition most favorable to a child's normal development. Exercise is an essential in the hygienic care which favors normal development, but a child who must be kept in bed in order not to overstrain a dilated heart before hypertrophy can take place, is debarred from all the usual forms of exercise. Besides the beneficial effects of exercise upon nutrition and development, another point must be considered. Any function deteriorates when not called upon for use. The heart muscle needs exercise like other muscles. If no demand be made upon the heart during this long period, the heart cannot hypertrophy to meet increased and increasing demands, and when finally the patient is allowed out of bed, the strain upon the heart caused by the suddenly increased exertion, is likely to be more than can be successfully met by the muscular power of the organ.

The indication is to guard the heart from overstrain, but as developing muscular hypertrophy is gradually increasing the power of the heart, a gradually increasing demand, always kept within the limits of this power, is indicated.

These indications, to favor nutrition, and to gradually increase the burden placed upon the heart in order to prepare it for the activities of a child's life, can best be met by means of *massage*, and *graduated muscular exercise.* When all acute symptoms have subsided, gentle massage, preferably given by a trained masseur accustomed to cardiac cases, should be instituted, under the directions and watchful care of the physician. The treatment may be given daily, or at least twice a week. The amount of massage should be gradually increased. The physician should see the patient frequently immediately after the treatment, and should note whether it produces any symptoms of cardiac disturbance or any effect upon the pulse. This treatment is the best available substitute for exercise at this stage of the disease, and causes an improvement of the circulation, and of nutrition, in which the heart partakes.

The next step is that of exercise against resistance, given by a trained attendant, in which definite groups of muscles are systematically brought into action. These exercises must be slight at first, and must be carefully graduated, so that the increased demand made upon the heart is slow and steady. The massage is continued. The physician should keep the patient under close observation, examining him frequently after the treatment for any signs of relative overstrain. The amount of muscular exercise taken in this way is increased until the exertion is greater than that which would be made if the patient were up and about his room.

When this stage is reached, the patient begins to get out of bed and walk. The same system is pursued in regulating the amount of exertion permitted. I allow such patients at first to take only one or two steps, and gradually and steadily increase the amount of their activity, watching constantly for signs of excess. Thus the various gradations in which the child is allowed to walk about his room, to walk about that floor of the house, and finally to come down stairs, steadily follow. The further treatment of the case is carried out upon the same general principle, the details varying with the individual case. The idea is to increase by regular gradations the amount of exercise taken, until the child is leading the normal life of the fully compensated case of chronic endocarditis. I first permit walks of increasing length upon a level, and then let the patient begin on the stairs, going up one step more each day. Other forms of exercise are gradually added. Constant observation of the effect upon the heart is essential to the proper carrying out of this plan of treatment.

The other measures of treatment in this stage of chronic endocarditis are mainly those which tend to insure good nutrition and normal development. The diet and bowels must be carefully regulated, in order that no improper burden be placed upon the digestive powers. All the details which tend toward normal development, which are described under the Hygiene of Normal Children, must be carefully attended to. A quiet, well-regulated life, under the best possible hygienic conditions, is one of the most important factors in the establishment and maintenance of good cardiac compensation.

No cardiac stimulants or tonics are indicated in this stage of the disease. If the appetite is poor, nux vomica or some other bitter tonic may be given. Anemia must be combatted by the use of iron. If the diet is necessarily limited by any peculiar digestive idiosyncrasies, cod liver oil is often useful.

STAGE OF COMPLETE COMPENSATION.—With children in whom the only evidence of chronic endocarditis is the existence of the endocardial murmur and cardiac enlargement, therapeutic endeavor should be directed first at preventing disturbance of the heart from adverse influences, and second at fitting the child to encounter the demands of a useful adult life. The most important adverse influence in childhood is reinfection with rheumatic fever. The two measures of most importance in the prophylaxis of this disease are, first, removal of the tonsils, and second, a change of climate in the winter and spring months. I believe that tonsillectomy should be performed in every child having a compensated valvular lesion of rheumatic fever origin. The teeth, and all other possible foci of chronic infection, should receive proper care. Whenever possible, such patients should spend the winter and spring in a warm, dry climate, where a healthy, out-of-door life is practicable.

The other adverse influences which may interfere with cardiac compensation are intercurrent acute infection, faulty nutrition, and a relative excess of physical exertion. These can only be prevented by rigid scrutiny and regulation of every detail of the child's life and environment. The diet, education, exercise, sleep, fresh air, and all other hygienic details, should be carefully scrutinized. Special care should be taken to guard against the deleterious effect of overpressure at school upon general health. Special watchfulness is required at the age of puberty, because at this age there is a special liability to the development of disturbance of nutrition and anemia. Children grow very rapidly at this age, and often their rapid increase in height is at the expense of their general nutrition. Under these circumstances, overstrain of the heart is particularly likely to occur. Anemia must be combatted by appropriate medication.

The chief problem which confronts the physician in compensated valvular disease of the heart, is the proper regulation of the child's exercise. On the one hand is the danger of disturbance or failure of compensation from relative over-exertion. On the other hand, no period is more valuable in fitting the child for the activities of adult life than the period of growth and development, and this training of the child to meet adequately the demands of an active life can only be brought about by accustoming the developing heart to cope with physical exertion without disturbance of function. The possibility of overstrain from excessive exercise is a real one, but in my experience physicians are more apt to err on the side of safety, and to limit too much the amount of exercise permitted. The tendency is to regard overexertion rather than reinfection as the greatest danger which menaces the child. Consequently, when the diagnosis of an organic valvular lesion is established, exercise is too strictly limited. As a result, not only does the nutrition of the heart suffer with the nutrition of the child, but the heart itself suffers from lack of exercise. Children who, so to speak, are "put under a glass case" because they have heart disease, lose the benefits to be gained in the precious period of growth and development, and enter adult life unfitted to cope with the demands which will inevitably be made upon the functional power of the heart.

The child with compensated valvular disease should be permitted and encouraged to devote fully as much time to exercise as the normal child. Only the character of the exercise must be modified. All competitive games and exercises which entail a prolonged and severe strain should be forbidden. These include football, baseball, tennis, competitive running, swimming, and mountain climbing. On the other hand, walking, skating, horseback riding, rowing, bicycling on the level, and regulated gymnastics should be encouraged in moderation. These can only be general rules. Each child must be carefully watched, and the amount and character of its regular systematic exercise planned and regulated to meet the peculiarities of the individual case. If the effect of exercise be carefully watched, its amount may be gradually increased until the heart is trained to encounter all the demands of an ordinarily active life without overstrain.

STAGE OF PARTIAL COMPENSATION.—When symptoms of disturbance of compensation are present, it is important that the morning and evening temperature be taken, in order to make sure that the cardiac insufficiency is not due to reinfection. If there is any fever, the child should at once be put to bed and treated as a case of acute endocarditis. If no temperature be present, the physician should conclude that the symptoms are caused by relative overload. He is now confronted by the decision whether the child should be put to bed, or whether it should continue to try to lead a more active life. The dangers of physical activity on the one hand, and the disadvantages of unnecessary rest treatment on the other, have already been described. The decision must depend upon the severity of the symptoms, and upon whether or not their severity is increasing. With severe or increasing symptoms, the child should be put to bed until they have disappeared, and then allowed to resume a more active life gradually, under strict supervision. If the symptoms are mild, and not increasing, the amount of activity must be cut down until they are relieved, and every effort must be made to improve the child's general condition by hygienic regulation.

The cardiac tonics, particularly *digitalis*, are useful in cases of this character, given in doses appropriate to the age of the child.

Stage of Failure of Compensation.—Here also the first step should be the taking of the temperature, in order to exclude the possibility of acute endocarditis as the cause of the symptoms.

Cases of acute cardiac insufficiency, such as are comparatively common in adults, with sudden dyspnea, cyanosis, irregular action of the heart or gallop rhythm, and a small, rapid pulse, are uncommon in children. Such cases are more often associated with arteriosclerosis and chronic myocarditis, lesions which children do not have. The treatment is that of adults, with hypodermics of camphor, caffein, and digitalis.

In the majority of instances the failure of compensation is more gradual. The first essential of treatment is *absolute rest in bed*, carried out in the manner which has been described in detail under acute endocarditis. As far as rest treatment is concerned, the treatment of cardiac insufficiency and its convalescence, with rest, massage, and later systematic exercises, is the same, whether the cause is acute endocarditis or failure of compensation.

The feeding of these cases is important. The venous stasis of the gastric mucosa often decidedly lowers the digestive power of children with broken compensation, and it is easy to produce indigestion from overfeeding. The starchy foods are particularly likely to be badly borne, and in the beginning milk diluted with lime water, white of egg, freshly-prepared beef juice, and whey should form the diet. Later zwieback or toast may be added, and then, if there is no digestive disturbance, the child can have meat and green vegetables. If nausea or vomiting develops, feeding should be omitted for twelve hours, and then the food should be given in smaller amounts at shorter intervals. The details of the diet must often be altered to meet the peculiarities of the individual case, the general principle being to nourish the child as well as possible without producing indigestion.

The question of restriction of fluid intake always arises in these cases. Theoretically the ingestion of a large quantity of fluid increases the burden thrown upon the heart. If edema be present, fluid is all the more contraindicated theoretically. Nevertheless, restriction of fluid with diminished renal elimination may cause toxic symptoms. I believe that restriction of fluid should not be used in cases without edema. When edema is present, a certain amount of restriction is advisable. The quantity of fluid allowed daily should never be less than the daily quantity of urine, and usually the giving of more liquid is necessary in order not to overburden the digestion with too much solid food.

There are certain medicines which strengthen the heart's action, and help to restore compensation. Among these *digitalis* takes the first rank. In general, digitalis is indicated whenever the heart's action is weakened to the degree of insufficiency, provided that the weakening is not due to an infection of the myocardium. In broken compensation, the drug is indicated when the effects of the weakening are clearly manifest. A certain number of cases in children begin to do well immediately when rest treatment is begun. In such cases, if improvement begins at once, and is continuous, digitalis need not be given. If the only symptom is shortness of breath, it is well to try the effect of rest alone before resorting to digitalis. If, however, there is cough, râles in the lungs, enlargement of the liver, or any edema of the skin, digitalis should be given. Palpitation, rapidity of action, and arhythmia are not in themselves indications for digitalis. There must be signs that the weakness of the cardiac action has resulted in the backing up of blood either in the pulmonary or systemic circulation.

The testing of the systolic pressure by means of the various forms of special apparatus which have been devised, is of comparatively little value in children. The conditions in which blood pressure examination is of the greatest aid in guiding the diagnosis are mainly those associated with arteriosclerosis, which are not seen in children. The indications and contraindications for digitalis in the cardiac diseases of children cannot depend upon blood pressure investigations. While I am not in any way decrying the enormous value of recent methods of diagnosis by means of the laboratory and of physiological apparatus, I must enter a warning. These methods, through their scientific exactness, have an undoubted attractiveness. On account of this, there is danger that in certain kinds of cases an exaggerated estimate may come to be placed upon their value, and that the value of the older methods of careful clinical observations and physical examination may be unduly minimized. The modern physician must be familiar with every physiological and laboratory method of proven clinical value, but must regard them, not as superseding older methods, but as valuable additions to his stock of weapons. He must know when to use them. In children, no matter what the blood pressure, and no matter what the particular valvular lesion, the only constant indication for the use of digitalis is cardiac insufficiency from chronic valvular disease.

The best indication of the effect of digitalis is the increase in the secretion of urine which follows its use. Its effect upon the symptoms of cardiac insufficiency should follow rapidly. When the doses of digitalis first given do not produce the desired effect, that is, when there is no slowing of the pulse rate, no increase in the secretion of urine, and no relief of symptoms, it is a sign that the doses are too small, and they should be cautiously increased until the desired effect is obtained. The signs that digitalis is being used in too large doses, or that its use is being continued too long, are the following: (1) Retardation of the pulse to a rate below that which is normal in a child of that age; (2) the appearance of the *pulsus bigeminus*, in which extrasystoles, that is, contractions of the ventricle without a preceding auricular beat, occur; (3) the appearance of the *pulsus alternans*, in which only every other ventricular contraction produces a pulse wave at the wrist.

Digitalis may be continued as long as it produces a notable effect without the signs just enumerated. When the edema and other signs of cardiac insufficiency have subsided, it may be omitted.

The preparations of digitalis most used in children are the *tincture* and the *infusion*. The tincture should be used as the routine preparation. The table of doses at the various ages given in Division II should be regarded as representing a minimum rather than a maximum. The proprietary preparation *digipuratum* has been widely recommended, and may be used in place of the tincture, if preferred. I have never observed any particular advantages over the tincture. If it be used, the doses may be arranged for the several ages in the same proportions to the adult dose as are those of the tincture. The *infusion* is believed by some to be more diuretic in its action, and to be better borne by children. I use it as a substitute for the tincture in cases in which there is an obstinate or resistant edema, or when the tincture fails to produce a satisfactory urinary secretion. The so-called active principles of digitalis have been isolated, and the preparation *digitalin* (Merck) may be used. Its only advantage is that it may be given hypodermically when the stomach is irritable. It should be used whenever digitalis is indicated, but cannot be retained by the stomach. The dose in children of from six to ten years is 1/120 to 1/60 of a grain every four hours, with careful watching, and in children of from ten to fourteen years, it is 1/60 to 1/40 of a grain.

Of the various substitutes for digitalis, none is so efficient. In children, however, *strophanthus* is sometimes better borne than is digitalis, and it should be used whenever the latter produces nausea, vomiting, or any untoward effect. The tincture should be used, in doses proportioned to the age of the child. Camphor, caffein, and theobromin are all cardiac stimulants, but cannot be considered effective substitutes for digitalis in chronic valvular disease with broken compensation. Their chief use will be considered under the treatment of special symptoms.

The principal symptom which requires special treatment in children is the *cardiac dropsy.* It is usually relieved by the digitalis, and a moderate restriction of the fluid intake. At times it may be resistant, and it is then a very difficult symptom to overcome. The first measure to try is the use of the saline cathartics, given early in the morning in concentrated form. Magnesium sulphate is the most effective. It is best not to use hot air baths, hot baths, or hot packs in cardiac cases in children. If the edema continues to resist digitalis, theobromin-sodium salicylate (diuretin) should be tried in addition, and continued as long as effective diuresis persists. Theocin may sometimes be tried after diuretin has lost its effect. Calomel is not useful as a diuretic in children. If these measures fail, caffein-sodium benzoate or salicylate may be tried for a time as a substitute for the digitalis. Persistent ascites or hydrothorax may be relieved by tapping.

For *sleeplessness* and *restlessness,* trional, either alone or combined with bromide, may be tried first. If the symptoms continue, opium should be used. Paregoric is the best form in which opium can be given to children, although severe cases may require hypodermics of morphin. While opium is theoretically contraindicated with a low output of urine, it must be remembered that it is the drug which next to digitalis has the most favorable effect upon the action of the heart. Children suffer a good deal from attacks of palpitation and of irritable heart. An ice-bag to the precordia may help these symptoms, but there is nothing like a little paregoric or morphin. I try not to use it when I am trying to get rid of a marked edema through the kidneys, but otherwise, it is my first choice for pain, palpitation, restlessness, sleeplessness, and severe dyspnea.

Vomiting can only be helped by giving nourishment in smaller amounts at more frequent intervals. If it becomes very severe, rectal feeding is indicated. Bromides given by rectum will sometimes be of service.

Cough is rarely severe enough to require special treatment. Paregoric will relieve it.

Anemia should be combatted by the usual methods.

CHRONIC ADHESIVE PERICARDITIS

(Adherent Pericardium)

ETIOLOGY.—The cause of this condition is a foregoing inflammation of the pericardium. The etiology is therefore the same as that of acute pericarditis. The commonest inflammation leading to adherent pericardium in children is that caused by rheumatic fever. The only other important inflammation causing the condition in early life is that of tuberculosis. The acute pericarditis caused by the extension of a pneumococcus or streptococcus infection from the pleura or other adjoining tissues, is usually fatal in the acute stage, but occasionally the acute process may subside, leaving chronic adhesions.

In the description of acute pericarditis it was stated that recovery is rarely if ever complete in the anatomical sense. The exudate which is not absorbed undergoes fibrous tissue organization, which causes adhesions between the visceral and parietal layers of the pericardium. In the majority of cases these adhesions do not interfere with the action of the heart enough to cause symptoms. In certain cases, however, the result is a marked disturbance of the circulation.

PATHOLOGICAL ANATOMY.—The fibrous tissue adhesions between the layers of the pericardium may be only partial, confined to limited areas of varying extent, or may be general, completely obliterating the pericardial cavity. When the chronic inflammation has not extended to the tissues outside the pericardial sac, and when there is little production of new fibrous tissue, the adhesions do not usually produce symptoms, even when there is a complete obliteration of the cavity. Adherent pericardium without symptoms during life is a not uncommon autopsy finding. At times the chronic inflammation extends to the tissues of the mediastinum, resulting in the formation of fibrous tissue bands joining the fibrous pericardium to the thoracic wall in front, to the mediastinum behind, and to the central aponeurosis of the diaphragm below. Sometimes all the mediastinal tissues are converted into a dense fibrous mass, firmly bound to all the adjoining structures. When these fibrous adhesions outside the pericardium are few and not firm, they interfere little if any with the action of the heart. When there are numerous and firm adhesions to the surrounding structures, there is marked interference with the action of the heart. In children, in whom there is little or no tendency toward chronic myocarditis, the chief result of this interference is cardiac hypertrophy, and if there be relative overload, there is dilatation and cardiac insufficiency. Valvular lesions almost invariably are also present in cases of rheumatic fever origin.

There is further disturbance of the circulation in these marked cases of adhesive pericarditis, from traction upon the great vessels. Traction on the aorta increases the work of the left ventricle, while traction on the pulmonary veins or venae cavae causes congestion of the pulmonary or systemic circulation. In such cases there may occur a widespread proliferative inflammatory thickening of the serous membranes, pleura or peritoneum. There may be proliferative fibrous thickening of the capsules of some of the abdominal viscera, particularly the liver, causing the so-called "chronic universal perihepatitis."

SYMPTOMS.—In a certain number of cases there are no symptoms during life in any way peculiar to the pathological condition found post-mortem. In most cases having symptoms, the clinical manifestations are those of disturbance of the circulation from cardiac insufficiency. Among them may be numbered palpitation, tumultuous action of the heart, precordial oppression, dyspnea, anxiety, syncope, and sometimes cyanosis. There may be cough, enlargement and tenderness of the liver, and edema. These symptoms are practically identical with those of uncompensated chronic valvular disease, and as lesions of the heart valves are usually also present, there may be nothing in the history of the case especially suggesting adhesive pericarditis. Whenever there is a history suggesting a previous acute pericarditis, or whenever the symptoms of cardiac insufficiency seem unduly prolonged and resistant, the possibility of adherent perirardium should be suspected. In children with this lesion, the restoration of compensation through rest and digitalis is often difficult and sometimes impossible. So long as they remain quiet in bed they are comparatively free from symptoms, but the least exertion causes dyspnea, and if any effort is made to get them up and about, the symptoms of marked cardiac insufficiency return at once. Progressive increasing circulatory weakness in a child with chronic endocarditis, without any intercurrent acute attack, is always suggestive of pericardial adhesions.

In a certain number of cases, in which there is a marked chronic mediastinitis involving the region of the diaphragm, and extending to the peritoneum and capsule of the liver, the symptoms resemble those of cirrhosis of the liver, ascites being the most prominent clinical manifestation.

It will be seen from this description, that the diagnosis of adhesive pericarditis must depend mainly upon the physical examination.

PHYSICAL SIGNS.—There are no characteristic physical signs which are constantly present in adhesive pericarditis. The most important is *systolic retraction* in the region of the lower part of the precordial area. This may be limited to one or more intercostal spaces in the area of the cardiac impulse, and if so, it is only suspicious but not diagnostic, as it is sometimes seen with pleural adhesions at the margin of the left lung, and occasionally without any adhesions. At times the systolic retraction involves the epigastrium, the costal cartilages, and even the lower end of the sternum. In such a case, the sign is of much greater diagnostic significance, but is still not absolutely pathognomonic.

The other signs seen on inspection are either only occasionally present, or else are not pathognomonic. A marked undulatory movement of the precordia is sometimes seen in adhesive pericarditis, but may occur in dilatation without adhesions. Immobility of the diaphragm, or a marked lessening of its respiratory movement, is seen at times. Systolic retraction of the tenth and eleventh intercostal spaces below the left (and rarely also the right) scapula—Broadbent's sign—is sometimes seen, and when present, is considered diagnostic. Diastolic collapse of the veins of the neck has been regarded as an important sign by some writers, but is not often present. Other visible signs described from time to time are of little importance.

On palpation there is often enfeeblement, and sometimes disappearance of the cardiac impulse, but this is not pathognomonic. A palpable diastolic shock has been described in some cases. The pulsus paradoxus in which the radial pulse grows weaker with inspiration, is sometimes present, but is of diagnostic value only when there is a concomitant inspiratory engorgement of the jugular veins (Sahli).

On percussion there is always an increase in the area of cardiac dulness. The area usually corresponds to that of a hypertrophied or dilated heart, and when, as is usual, valvular lesions are also present, the evidences of cardiac enlargement have no diagnostic significance. Rarely adhesive pericarditis is the sole cardiac lesion, and in such a case, marked cardiac enlargement without an endocardial murmur is strongly suggestive. Occasionally there is dulness in the fifth and sixth right interspaces, with an obtuse cardio-hepatic angle.

On auscultation, the heart sounds may or may not be weakened. There are sometimes fine friction râles of a parchment-like character audible at the margins of the area of superficial cardiac dulness. If they persist when the breath is held, they are strong evidence of pleuro-pericardial adhesions. Various other peculiar sounds are heard in rare cases.

In cases with perihepatitis, *ascites* is the most prominent symptom. It is usually very marked. Edema of the lower extremities may follow later. The liver is usually enlarged.

DIAGNOSIS.—The lack of constant characteristic clinical evidence renders the diagnosis of chronic adhesive pericarditis difficult. There are some cases in which the signs are so typical, or there is such a conjunction of suggestive signs, that a diagnosis can be made with a fair degree of certainty. In other cases the evidence is less positive, and only a probability or possibility of adherent pericardium can be established.

In differential diagnosis, the two conditions to be considered are chronic endocarditis and cirrhosis of the liver. Chronic endocarditis is present in most cases, and the only question is whether adhesive pericarditis is present as an additional lesion. Adherent pericardium is distinguished from cirrhosis of the liver only by the presence of physical signs characteristic of the former disease.

PROGNOSIS.—In children in whom chronic adhesive pericarditis and chronic mediastinitis are sufficiently marked to interfere with the action of the heart, or in whom the process is sufficiently extensive to cause perihepatitis and ascites, the prognosis is unfavorable. Such patients may live for months, or even years, but usually the additional burden imposed upon the circulatory apparatus eventually leads to a fatal ending.

TREATMENT.—This is purely symptomatic, and is the same as the treatment of chronic valvular disease with broken compensation, namely, rest and digitalis. The digestive system usually requires particular attention, as it is apt to be much disturbed by the results of venous stasis. The saline cathartics are useful in these cases, with an occasional administration of calomel. Cases with marked ascites must be relieved by frequent tapping.

Brauer has carried out an operative treatment for the relief of this condition, which he calls "cardiolysis." It consists in resection of the ribs and cartilages, and even of the margin of the sternum, to which the heart and pericardium are adherent. This converts the resistant chest wall into a soft, yielding tissue, which permits better contraction of the heart and better movements of the diaphragm. Good results have been obtained from this operation.

III. FUNCTIONAL DISTURBANCES OF THE HEART

Disturbances of the function of the heart unconnected with organic disease, but due to the multiple causes which may produce various derangements of function in any important physiological system of the body, are not uncommon in children.

ETIOLOGY.—The essential mechanism of the action of the heart is nervous and muscular. The nervous mechanism may be deranged by a great number of general causes which disturb the function of the nervous system. In children the nervous system is relatively undeveloped, and is particularly liable to excessive excitation from comparatively slight causes. This condition of increased nervous excitability is so characteristic of early life, that it may almost be considered as normal, and the difficulty in describing the functional cardiac disturbances lies in drawing the line between the normal and the abnormal.

From a practical point of view, it is best to classify as abnormal functional disturbances, only those conditions in which symptoms are present. By symptoms I here mean either symptoms complained of by the patient, such as palpitation, or some peculiarity of the child's reaction to its surroundings and daily life observed by the parents, such as, for instance, attacks of syncope. I do not mean some peculiarity of the action of the heart, such as tachycardia or arhythmia, only observed by chance by the physician in the course of a routine examination, and unaccompanied by subjective symptoms.

The origin of an abnormal disturbance of the nervous mechanism of the heart is mainly either *reflex* or *toxic.* The causes of reflex disturbance are the general conditions which lead to nervous hyperexcitability, the various violations of the proper hygiene of the nervous system in early life, such as excessive mental stimulation, over pressure at school, faulty methods of discipline, and emotional disturbances such as fright or excitement. There may be specific sources of reflex stimulation, such as disturbances of digestion. Causes of toxic disturbance are the excessive use of tea or coffee, cigarette smoking, and again, certain disturbances of digestion and metabolism.

The origin of abnormal disturbance of the muscular mechanism of the heart is chiefly developmental or nutritional. The commonest cause is an inequality in physical development often seen in children, through which the growth and development of one part of the body is more rapid than that of another. This is particularly common about the age of puberty, when children grow very rapidly. If the rate of growth of the rest of the body is more rapid than that of the heart, the result is an essential muscular insufficiency of the heart, not due to organic disease, but marked enough to produce symptoms. In anemia, and in other disorders in which the general nutrition suffers, that of the heart suffers also, and the result is again a muscular insufficiency.

SYMPTOMS.—The principal symptoms of functional disturbance of the heart in children are attacks of palpitation, tachycardia, or arhythmia.

In an attack of *palpitation*, the child complains of violent action of the heart, and sometimes of a sense of precordial oppression. On physical examination, tachycardia is usually found, and sometime slight arhythmia. There is usually a marked visible pulsation of the carotids. Sometimes headache or dizziness is complained of. The duration of the attack varies from a few minutes to a number of hours.

Tachycardia without other symptoms occurs in children of a nervous type or who are not in the best physical condition, from such slight causes that it can hardly be regarded as a manifestation of disease. It is best never to attach any serious significance to tachycardia as the sole symptom, as in some cases a very rapid pulse must be considered an idiosyncrasy. Gràve's disease should not be considered, unless there is also exophthalmos or goitre. It is notable that in these attacks of tachycardia, which may persist for days or weeks, the pulse rate is much slower during sleep, and may be quite normal.

There is a rare form of tachycardia seen in children, which is much more severe, and which is called *paroxysmal tachycardia*. In this condition there suddenly develops an extremely rapid pulse rate, which may reach 200 or more per minute. The attack may last only a few minutes, or may persist for days or weeks. The pulse is often abnormally slow after an attack. When the attack is prolonged, dilatation of the heart with a systolic murmur may develop, and may lead later to the symptoms of cardiac insufficiency. The etiology of this condition is obscure; it is probably toxic, due to some disturbance of metabolism.

Arhythmia is so common in early life as an indication of a relatively undeveloped heart-regulating mechanism, that in itself it cannot be considered an important functional disturbance. It is very often seen in children, who in all other respects are perfectly well, especially in the earlier years of childhood. The usual type is that which is called sinus arhythmia, reflex arhythmia, or vagus irregularity. The cause is the reflex stimuli which are constantly reaching the heart, and which in the normal adult are insufficient to disturb the regulatory mechanism. In children the mechanism is more easily disturbed, on account of the relatively undeveloped nervous control of function. The result is a variation in the rate of the heart. As shown by Einthoven, stimuli pass up the vagus nerves with every respiration. The irregularity of the heart is often rhythmical, corresponding with respiration, the rate increasing with inspiration and diminishing with expiration. In other cases there are irregular retardations or pauses. When this condition is unassociated with any subjective symptoms, it may be ignored.

Another type of arhythmia seen in children is due to *extra systoles.* In this condition a second, somewhat weaker, systolic contraction occasionally immediately follows the first, giving two beats close together. Sometimes after an extra systole the next beat occurs "on time", but more often there is a long diastolic pause, the next regular beat being omitted. The extra systole may not be strong enough to reach the wrist, and in such a case palpation of the radial pulse only shows the long pause if it is present. The extra systole can, however, be perceived by auscultation.

The mechanism of extra systole is an abnormal stimulus arising in the auricle or ventricle. When it occurs without subjective symptoms, it should be ignored. When it is noticed by the patient, or when it accompanies any subjective symptoms such as palpitation or precordial oppression, it should be regarded as a manifestation of disturbed cardiac function from one of the causes enumerated under etiology.

DIAGNOSIS.—The only problem in the diagnosis of functional disturbance of the heart, is the exclusion of organic disease. As the only important organic diseases of the heart encountered in early life are those due to congenital malformations or to infection, the problem of their exclusion is much easier in children than in adults. In a child having any symptoms referable to the heart, the diagnosis does not depend upon electrocardiographic tracings, but upon the results of a careful physical examination. If the characteristic murmurs, cardiac enlargement, and other signs of organic disease are not present, we can conclude that the cause of the symptoms is a functional disturbance.

The only difficulty in diagnosis is caused by the frequent occurrence of *accidental murmurs* in early life. In order that the distinction between these murmurs and those caused by organic disease should be emphasized, they have been often referred to as "functional murmurs." There is no evidence, however, that they are connected in any way with any especial disturbance of cardiac function. They may be present in cases with functional disturbance, just as they may be present in normal children. When present in cases of functional disturbance, their chief significance is that they complicate the diagnosis, by suggesting the possibility that the symptoms are due to organic disease, particularly mitral insufficiency. The absence of cardiac enlargement, of accentuation of the pulmonic second sound, and of transmission of the murmur into the axilla, are the chief points in the recognition of the accidental character of these murmurs.

PROGNOSIS.—As the etiology of functional disturbance of the heart is relative underdevelopment plus causes acting from outside the body which are removable, the prognosis is wholly favorable. The promptness of recovery depends upon the cause, and the ease with which it can be recognized and removed.

TREATMENT.—The treatment consists in the removal of all the possible causes acting from the outside. Such causes as cigarettes, tea, or coffee, are easily removed. Other causes can be removed only as a result of a most minute inquiry into every detail of the patient's life and environment. Careful hygienic regulation is then indicated. In the cases occurring at puberty, due to rapid growth of the body, and relative muscular insufficiency of the heart, exercise must be carefully regulated, and every effort must be made to insure the best possible nutrition. Sometimes the giving of crackers and milk between meals works well in these cases. Anemia should be combatted with iron.

In severe cases with palpitation or other subjective symptoms, the bromides are sometimes useful in the attacks, during the period before the general hygienic treatment begins to take effect. In paroxysmal tachycardia, complete rest, nervous sedatives, and digitalis are indicated.

IV. DISEASES OF THE BLOOD VESSELS

In children, disease of the blood vessels is so uncommon as to be relatively unimportant. The most important arterial diseases of adult life, namely aneurism and atheroma, are extremely rare in childhood. Still, a number of cases of these affections have been reported, sufficient to show that children are not entirely exempt. Such cases, however, must be regarded as pathological curiosities rather than conditions with which the physician must be prepared to encounter and recognize. Their pathology, symptomatology, diagnosis, and treatment present no essential peculiarities in childhood, and will not be described here.

The commonest affections of the blood vessels encountered in early life are thrombosis and embolism; these lesions are not peculiar to childhood. The principal affections of the blood vessels peculiar to early life are coarctation of the arch of the aorta, and arterial hypoplasia.

Coarctation of the Arch of the Aorta.—This is a rare congenital lesion of developmental origin. It consists in a constriction or a complete occlusion of the aorta near the point of entrance of the ductus arteriosus. In all cases with complete occlusion, and in some with partial constriction, the ductus arteriosus remains patent. The result of this lesion is the establishment of a more or less complete collateral circulation through the upper intercostal and mammary arteries above, and the lower intercostal and epigastric arteries below. When the constriction is above the open ductus, the blood in the collateral circulation flows from below upward, and when it is below the open ductus, the flow is from above downward. In consequence of the completeness of the collateral circulation, there may be no symptoms, and there are instances on record where patients with this malformation have lived until old age. Usually, however, growth and nutrition suffer, and there may be dyspnea.

On physical examination the enlarged vessels which carry on the collateral circulation may show superficially. There may be a notable difference in the intensity of the radial and the femoral pulse respectively. There may be visible pulsation and palpable systolic thrill in the suprasternal notch. Sometimes a loud systolic murmur is heard in the second and third left interspaces.

Death may occur from circulatory failure, or even from rupture of the heart or aorta, but is usually due to intercurrent disease.

Arterial Hypoplasia.—This also is a rare congenital developmental anomaly. The aorta, and sometimes the large arteries have a caliber much less than the normal. The chief clinical manifestation is an interference with growth and development. A positive diagnosis cannot be made during life.

THROMBOSIS.—This occurs in children from the same causes as in adults. The most common situation is the cranial sinuses, but thrombi may form in any of the large vascular trunks. The symptoms produced by thrombosis will be described in connection with the diseases of the various parts of the body affected.

EMBOLISM.—This is rare in early life, even in acute endocarditis. The symptoms produced depend upon the situation of the vessels occluded by the emboli, and upon whether or not the emboli are infected. Emboli produce symptoms referable to the organ or part of the body supplied by the occluded vessel. When infected, they cause abscesses.

DIVISION X

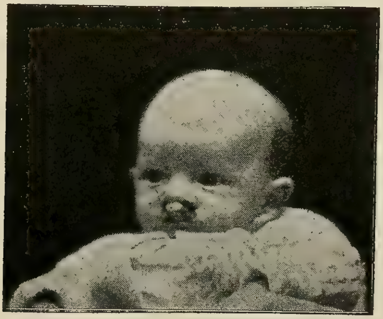
DISEASES OF THE MOUTH, NOSE, PHARYNX AND EAR

I. DISEASES OF THE MOUTH

MALFORMATIONS

HARELIP AND CLEFT PALATE.—These are the commonest congenital malformations of the mouth. If the maxillary process on one or both sides of the face fails to unite with the intermaxillary process, a cleft will remain open in the contour of the upper lip on one or both sides of the intermaxillary bone, and hence we shall have single or double harelip as the case may be. If the cleft extends the whole distance from mouth to nostril it is called complete, but if the nostril is not reached by the opening it is called partial harelip. If there is a failure of the palatine processes to join, one or both nostrils will open into the roof of the mouth as well as into the pharynx, and we shall have the malformation known as cleft palate. This may be a large chasm running the whole length of the roof of the mouth, or may be only a small opening, or nothing but a bifurcation of the tip of the uvula may be left to show that the normal process of development has not gone on to completion.

FIG. 224



Double harelip

Besides their unsightly appearance, which always causes the mother great concern, these malformations may so interfere with the infant's taking the nipple as to render sucking impossible. The difficulty of feeding, if the cleft involves the hard as well as the soft palate, is very great, and can best be accomplished with a spoon or medicine dropper. In some cases, with feeble infants, it is impossible to get the infant to take enough milk with the spoon or dropper, and gavage may be employed with advantage. The greatest care must be taken to keep the infant's mouth as clean as possible, by gentle washing with warm water after every feeding. Care must be taken not to injure the epithelium, and nothing more irritating than a camel's hair brush must be used.

The treatment of harelip and cleft palate is surgical. The time of operation should be left to the judgment of the surgeon. At times a deadlock occurs, the surgeon wishing to wait until the infant's general condition is better, while the physician wishes the operation performed soon, to remedy a condition which is injuring the infant's general condition by serious interference with its nutrition. In such a case the physician should make clear to the surgeon the difficulty of improving the nutrition while the malformation exists. In my experience, cases of harelip do best when operated on as early as possible after nutrition has become established. This means in the early days of life with normal vigorous babies, and in the second month with feeble or delicate babies. Cases of cleft palate do best when operated on in the second year, unless the physician finds satisfactory nutrition impossible during the first year.

TONGUE-TIE.—In quite a number of cases the frenum of the tongue is abnormally short at birth. In extreme cases the tip of the tongue is so closely bound to the lower jaw that it cannot be protruded beyond the line of the gum or touched to the roof of the mouth. The mother usually notices that the infant does not nurse readily, and brings it to the physician to discover the cause. In most cases on passing the finger into its mouth the infant is found to suck fairly well; but there can be no doubt that this condition, which is called tongue-tie, interferes somewhat with the process of sucking.

The treatment is to cut the frenum. This operation should be followed by no hemorrhage and requires no dressing. With the child's head held in a fairly good light by an assistant, and the lower part of the tongue guarded with the perforated flange of a director, a small cut is made in the tense frenum with a pair of blunt-pointed scissors. By making the cut close to the gum there is no danger of wounding the ranine artery. The cut is prolonged as far as is necessary by tearing with the finger-nail.

Children who have not learned to talk at the usual time in the second and third years are frequently believed to be tongue-tied, and the parents wish the condition to be treated. Large numbers of children are taken to the physician under this supposition but in very few instances are they tongue-tied. They are really examples of retarded speech.

RARE MALFORMATIONS. There are a certain number of rare malformations seen involving the cavity of the mouth. Among them are *macroglossia*, or congenital hypertrophy of the tongue, *microglossia*, or congenital arrested development of the tongue, and *bifid tongue*. *Bifid uvula* is not uncommon in connection with cleft palate, and is occasionally seen alone.

STOMATITIS

Diseases of the mouth occur more frequently and in much greater variety in infancy and early childhood than at any later period of life. The undeveloped salivary function seen in the earliest months of infancy may be a contributing cause to the frequency of disease of the mouth. The most important cause however, is the frequency with which the mouth is exposed to irritation from without. The mucous membrane of the mouth during the greater part of the period of infancy is subject to external sources of irritation to which older children are not liable. During the first year there is more or less mechanical irritation through the mechanism of sucking. The contents of the feeding-bottle may be overheated, causing thermal irritation. Foreign bodies are constantly introduced into the mouth by the infant itself, and sometimes by parents when they insert their fingers to feel for a tooth.

The microörganisms of the mouth are very numerous, and they have been definitely associated with some forms of stomatitis. It is not known what part they play, if any, in producing other forms.

As in many other diseased conditions, there is no close or necessary correspondence between the cause and the lesions seen in stomatitis. Classification on a strictly etiologic basis is not possible, and the various conditions must be differentiated and described mainly on the basis of the lesions found. In some cases the lesion is very definite, but the cause is wholly unknown. In other cases, the same lesion is produced by a variety of causes, or the same cause produces a variety of lesions.

The following is a summary of the principal etiologic factors in stomatitis, with the lesions produced by each one.

1. *Mechanical, chemical, or thermal injury*. The lesion produced is usually a catarrhal stomatitis.

2. *Hemorrhage* (in Scorbutus).—The lesion produced is an *ulcerative stomatitis*.

3. *Toxins* from within, such as the *mineral poisons*.—The lesion is an ulcerative stomatitis.

4. *Infection*.—The mucous membrane of the mouth may show an

efflorescence, which is only part of the general efflorescence of an infectious exanthem. A specific infection causes the lesion known as *thrush.* Various bacterial infections may produce lesions in the mouth which take the forms either of *ulcerative*, of *psuedomembranous*, or of *gangrenous stomatitis.*

5. *Unknown cause.*—There is a definite disease, usually known as stomatitis herpetica, of which the cause is wholly unknown.

STOMATITIS CATARRHALIS

ETIOLOGY.—The condition is usually of traumatic origin. The causes may be mechanical, thermal or chemical.

Mechanical.—Among the most common mechanical causes may be cited the irritation produced by rubber nipples, too vigorous cleansing of the mouth, injudicious rubbing of the gums during dentition, and local irritation from a tooth.

Thermal.—The thermal form of traumatism may result from the administration of food which is too hot.

Chemical.—The chemical irritation may arise in various ways, as from lack of cleanliness in the mouth, with its resulting fermentation, and from the elimination of irritating products from the salivary glands, apparently connected in some way with disturbance in the gastro-enteric tract.

SYMPTOMS.—The most marked symptom of stomatitis is pain. The infant is restless, usually has a slightly heightened temperature, and refuses to take its nourishment. The lesion is essentially an inflammatory one, and occurs in different grades. On examining the mucous membrane in these cases, it is seen that the entire lining of the mouth is intensely reddened, that the temperature of the mouth is increased, that there is usually a certain amount of swelling, and that, although the mucous membrane may be dry under certain circumstances, especially at first, yet, as a rule, there is later a hypersecretion of mucus and saliva. When the catarrhal condition is at its height the mucous membrane is so vulnerable that even slight traumatisms may cause abrasions. The saliva is acid in its reaction, and when secreted in large quantities flows out of the mouth upon the chin and neck, sometimes causing considerable irritation. The tongue is dry and white at first, then becomes of a grayish color, and as the secretion of saliva increases, the coating of the tongue is washed off and its surface becomes red.

PROGNOSIS.—The prognosis of stomatitis catarrhalis is, as a rule, good. Although the disease does not run a definite course, yet in most cases after a few days the pathological condition improves and the symptoms grow less severe. The course of the disease is, however, often lengthened by the secondary conditions which arise from the gastric disturbances which may be caused by swallowing the irritating secretions of the mouth. In weak, poorly nourished infants who refuse to nurse or take the food which is given them, serious results may arise from a lack of sufficient nourishment, so that in these cases the prognosis is always grave. In older children the prognosis is very good.

TREATMENT.—Although stomatitis catarrhalis may run a favorable course without any treatment whatever, yet there are so many causes which may prolong its course or give rise to secondary affections that it is exceedingly important to treat the disease at once. The indications for treatment are to relieve the pain and to allay the irritation of the mucous membrane so that a sufficient amount of nourishment may be taken by the infant to prevent it from being harmed by a lack of nourishment or by a secondary disturbance of the gastro-enteric tract. If the cause can be ascertained, it should be removed at once. The local application of a one to two per cent cold solution of bicarbonate or borate of sodium in distilled water is indicated. This solution should be used very gently every half-hour when the infant is awake, by means of a dropper, and occasionally on a clean swab of absorbent cotton. The infant should be systematically fed at regular intervals, whether it resists or not; and if it will not suck from the nipple, its milk should be administered with a spoon or dropper. Breast milk may be obtained by pumping.

When the stomatitis proves to be intractable and lasts for more than three or four days, the mouth can be gently touched with a cotton swab wet with a one per cent solution of nitrate of silver. This should be done once a day, and the mouth washed carefully with cold sterilized water after the application.

When there are any abrasions which show a tendency to extend or to form an ulcer, they should be touched with a little nitrate of silver melted on the end of a silver probe. These abrasions are often so painful that in themselves they may prevent the child from taking its food, and after they have been treated with the nitrate of silver the child will often again take its nourishment readily.

STOMATITIS HERPETICA

(Aphthous Stomatitis; Vesicular Stomatitis; Follicular Stomatitis)

The name herpetica has been adopted for the next form of stomatitis, because it seems to describe most nearly the lesion which is seen on the mucous membrane, although it is not definitely settled that it is a true herpes.

The disease consists of a catarrhal stomatitis in the course of which certain lesions resembling sub-epithelial vesicles surrounded by areolae occur irregularly and in different parts of the entire buccal cavity.

ETIOLOGY.—No cause, either local or general, has as yet been determined for this disease. Various microörganisms have been observed in the mouth when it is affected by stomatitis herpetica, but no actual connection has been discovered between them and the disease. This affection may be found associated with a number of other diseases, but usually occurs alone. It does not seem to be contagious, nor to be especially connected with diseases of the gastroenteric tract or with dentition, although it very commonly occurs during the dental period. It appears to be the result of certain toxic influences which act upon the peripheral nervous system and produce an herpetic efflorescence on the mucous membrane which corresponds closely to that which is seen in herpes on the skin.

The disease is not common in the first year of life, but is frequently seen in the earlier years of childhood.

SYMPTOMS.—In addition to the usual lesions of a stomatitis catarrhalis, spots, not necessarily symmetrical or unilateral, of different sizes and of different shades of white or grayish white, appear in various parts of the mouth, especially on the inner surface of the lip, on the side and under surface of the tongue, and on the gums. These lesions do not affect the follicles of the mouth, and the efflorescence cannot be called follicular, as it is closely connected with the muciparous glands. The lesions make their appearance with great rapidity, and develop very quickly from a macule into a vesicle. The action of the secretions of the mouth upon these lesions necessarily prevents them from having the same vesicular appearance that they would present on the skin. The vesicles are quickly broken, their covering being washed away, and shallow circular ulcers are found, which come in successive crops. These ulcers are so shallow, that the appearance is suggestive of an exudation on the mucous membrane rather than an excavation within it. They are of a light yellow color, and the small new ones have a red areola. The older ulcers are grayish, and their appearance may suggest a false membrane. The ulcers are about one-eighth of an inch in diameter, but a number may coalesce to form large patches. Their number is very variable.

An infant or young child affected by stomatitis herpetica presents a very characteristic appearance. It looks dull and apathetic, and wishes to lie quietly in bed. It usually has a heightened temperature, and evidently suffers pain and heat in its mouth. The saliva flows from the mouth in large quantities, and often irritates the chin and neck to such an extent that an eczematous condition results. The child refuses to take its nourishment, and is very fretful and restless.

These symptoms continue for four or five days or a week, and sometimes extend over a period of two weeks, the disease then disappearing of itself; in fact, it appears to be self-limited. Unless the lesions of stomatitis herpetica are complicated by those of stomatitis ulcerosa, the saliva is never fetid.

PROGNOSIS.—In general the prognosis is favorable. Feeding is always difficult, and in feeble or delicate infants, the problem of nutrition may become very serious.

TREATMENT.—It is usually stated that no internal treatment is of benefit in stomatitis herpetica. In my experience the course of the disease may very often be shortened by the internal administration of chlorate of potassium. To a child in the first year, one grain may be given every hour during the first day, and every two hours thereafter. In the second year one and a half grains may be given each hour, and in children over two years, the dose should be two grains. Potassium chlorate is rapidly excreted in the saliva, and thus is kept in constant contact with the lesions of the disease.

The local treatment of the mouth, and the management of the feeding, should be carried out in the same way as for catarrhal stomatitis.

THRUSH

(Sprue; Soor; Muguet)

(Stomatitis Mycosa; Stomatitis Hyphomycetica)

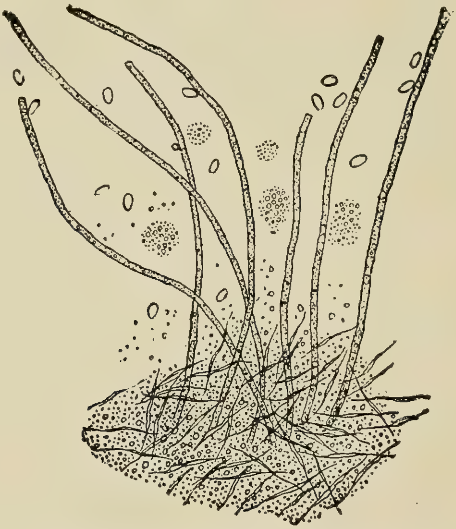
Thrush is a form of stomatitis of parasitic origin, produced by the growth of a specific fungus.

ETIOLOGY.—The exact place of the thrush fungus in the classification of plant life has been the subject of much discussion, and has not been definitely settled. It was formerly supposed to be the *öidium albicans*, but this is now known not to be the case.

The fungus as seen under the microscope consists of delicate jointed threads (the mycelium) at the ends of which oval spores are developed. The spores of thrush are of wide distribution in nature, and contagion is not a necessary factor in the acquirement of the disease. Thrush develops with difficulty on a healthy mucous membrane, and infection is favored by anything lowering the protecting and resisting power of the epithelium. Catarrhal stomatitis is a very important predisposing cause. The injury to the epithelium coming from rough methods of cleansing the mouth, or from lack of cleanliness, is a common starting point. A dry condition of the mouth also leads easily to epithelial injury, and the scanty salivary secretion seen in the early months of life, or later in wasting diseases, is an important factor in explaining the frequency of thrush in young or atrophic infants. Infants with harelip, or other deformity of the mouth, are also particularly liable to thrush.

The disease may also be directly transmitted from another patient, by means of contaminated nipples, fingers, or implements used in cleansing the mouth. Thrush is very common in all infant asylums or institutions in which infants are in close contact, with neglect of cleanliness of mouths, bottles, nipples, and other implements.

FIG. 225

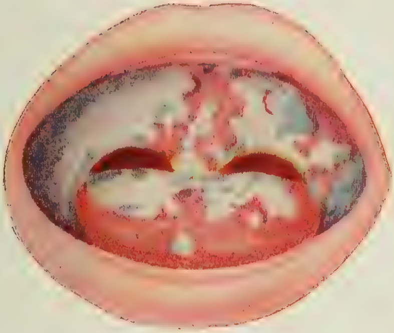


Mycelium of thrush interspersed with spores and fatty degenerated cells. (Low power Zeiss Oc. 3, Objective DD, glycerin)

PATHOLOGICAL ANATOMY.—The fungus of thrush may be found on any of the mucous membranes of the baby. The usual place for it to appear, however, is the mucous membrane of the mouth. The growth may take place on both squamous and cylindrical epithelium. According to Forchheimer, the first lodgement of the fungus comes between the epithelial cells of the mouth, and from this the growth works its way under the free surface of the mucous membrane. When directly on the free surface the growth is not so luxuriant and is principally in the mycelium form. In the case of a mucous membrane lined by flat or squamous epithelium, the growth is facilitated by the relation of the cells to one another. In a membrane lined by cylindrical epithelium, the growth takes place, but not so readily, because there is but one layer of the cells. After the first development the growth goes on very rapidly, and after it has formed a nidus the cells are pushed aside and are surrounded by mycelium, the whole presenting the characteristic appearance of thrush. The growth begins in small spots, sometimes

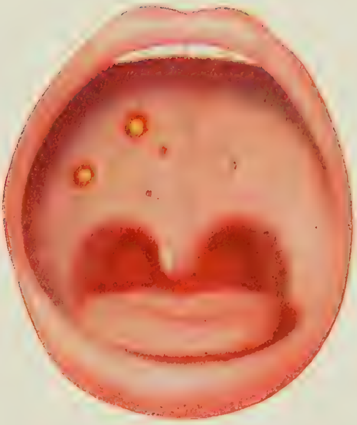
PLATE XII.

1



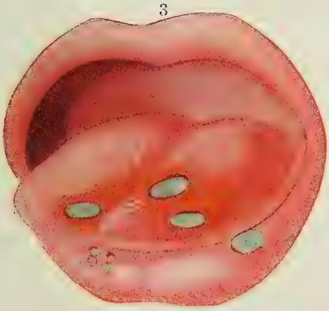
Thrush.

2



Varicella.

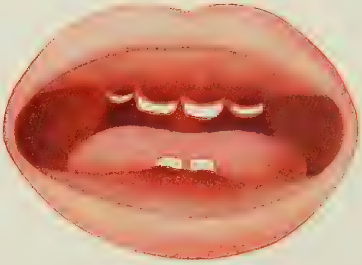
3



Stomatitis herpetica.

Aphthosa.)

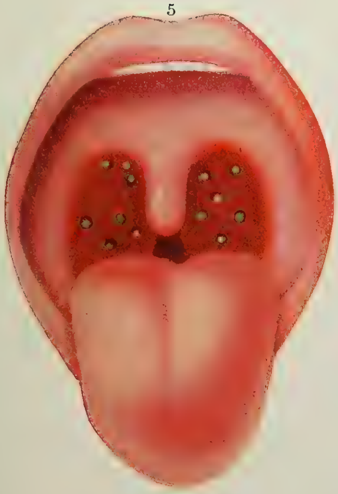
4



Stomatitis ulcerosa

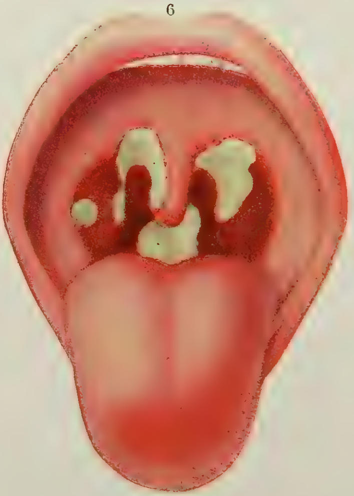
(Scorbutus.)

5



Follicular tonsillitis.

6

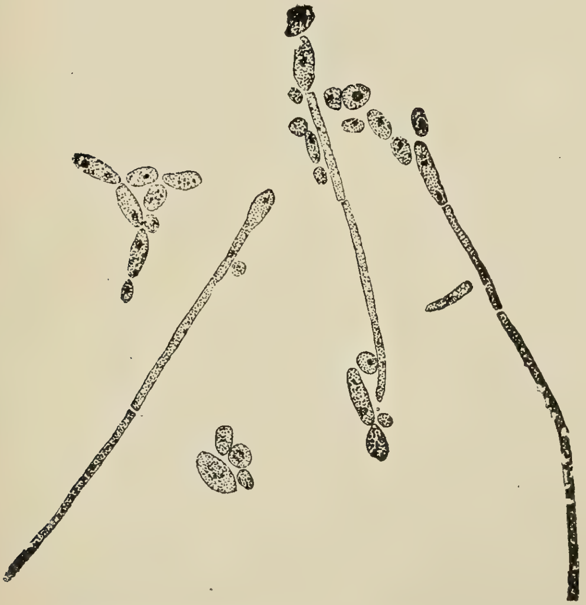


Diphtheria.

one, sometimes more, and at times the entire surface of the mucous membrane is covered with it. The fungus develops within the epithelium, and it requires considerable rubbing to remove the growth.

SYMPTOMS.—An attack of thrush usually begins with local symptoms of catarrhal stomatitis. At times, however, no symptoms are present, the fungus being the first abnormal condition which is noticed. The appearance of the fungus resembles closely that of curdled milk, though it is often of a grayish color. It does not look like a membranous exudation, but is raised in small patches above the level of the mucous membrane. The fungus usually develops on the inner borders of the lips, on the gums, on the tongue, and on the hard and the soft palate. It may extend to the tonsils and pharynx, and even into the esophagus. In the latter locality at times it has been found to grow so thickly that the lumen is almost entirely occluded. The local symptoms are commonly those of a mild catarrhal stomatitis. The general symptoms depend upon the extent of the local disease from which the infant is suffering. Infants affected with this disease soon become atrophic, from a lack of proper nourishment, as they are often unwilling to take their food or cannot swallow it without difficulty.

FIG. 226



Thrush showing the formation of spores in the mycelium. (Zeiss Oc. 3, homogen. immer. 2.0 mm)

DIAGNOSIS.—The differential diagnosis is seldom difficult to make. Curdled masses of milk on the inner surfaces of the lips and on the gums may resemble closely the fungus of thrush, but the former are easily wiped away, while the latter is difficult to dislodge. The nature of the disease is definitely determined by placing some of the growth under the microscope with a drop of liquor potassae, where it presents characteristic appearances.

PROGNOSIS.—The prognosis of thrush varies according to the general condition, the vitality, and the age of the subject on whom it is engrafted. The disease may last indefinitely if the mouth is not carefully treated, and its prolongation may render the prognosis more grave. When the growth is very extensive, as in the cases in which it has invaded the esophagus, the prognosis is very unfavorable. In these cases disturbances of the gastro-enteric tract are apt to arise and to increase the likelihood of a fatal issue. As a rule however, if the infant's health can be maintained, and if the local treatment is carried out thoroughly, the prognosis is favorable.

TREATMENT.—Thrush does not develop when proper attention is given to the cleanliness of the infant's mouth, and to nipples and bottles. The nipples and bottles should be boiled after each nursing, and nipples should be kept in a saturated solution of boric acid. If the baby is breast-fed, the mother's nipples should be washed with boric acid solution followed by boiled water, before each nursing.

The mouth, after each feeding, should be washed with a saturated solution of boric acid. The solution should be applied on absorbent cotton wrapped about the clean finger of the nurse, and the diseased parts should be gently rubbed. If the case is obstinately resistant, the diseased areas may be touched daily with a one per cent solution of formalin. When the disease is in the esophagus, it is best treated by the introduction of a soft rubber tube, which has been previously dipped in a saturated solution of boric acid.

The nutrition of the child must be attended to by careful regulation of the feeding. When sucking is painful, feeding with the spoon or medicine dropper may be employed; this procedure is often a good plan for a few days in beginning the treatment of any bottle-fed case, in order to avoid irritation of the mucous membrane from the rubber nipple.

STOMATITIS ULCEROSA

Ulcerative stomatitis is a pathological process of the mucous membrane of the mouth, occurring only when there are teeth, and characterized primarily by ulceration of the gum around the teeth, and secondarily by ulceration of other parts of the buccal mucous membrane.

ETIOLOGY.—The peculiar lesions of this disease are probably due to a specific infection. A typical bacteriological picture is constantly found in coverglass preparations made from the lesions. This picture shows the predominance of two kinds of bacteria, which are regularly associated; one is a large spindle-shaped bacillus, staining deeply with all the common aniline stains, called the *bacillus fusiformis*; the other is a long, delicate *spirocheta* which stains less deeply. The bacillus fusiformis has been cultivated as a strict anaerobe, and inoculations in animals have produced a local necrotic process.

The disease has occurred in epidemics, especially in asylums, but in general there is little evidence of transmission from one individual to another. The organisms are probably widely distributed, and frequently if not constantly present in the mouth. The same associated organisms are frequently found in carious teeth, and in ulcerative angina.

It is probable that predisposing causes are necessary to permit the organisms to produce the lesions of the disease. Caries of the teeth, neglected teeth, and unclean mouths, are probably the commonest antecedent conditions. Ulcerative stomatitis is very commonly seen in scorbutus, where the hemorrhage into the gum produces a lesion which forms a favorable focus for invasion by microörganisms. Bad surroundings, improper food, chronic malnutrition, and acute general infections may all at times play a part in lowering the vitality and resistance of the gums. The importance of the predisposing cause is shown in the fact that ulceration of the gums is often produced by such mineral poisons as mercury, lead and phosphorus, but even then the actual character of the ulcerative lesion is determined by whether or not the bacillus fusiformis and its associated spirocheta invade the injured tissue.

As the disease occurs only in children having teeth, it is not seen in early infancy. It is commonest in children having poor hygienic surroundings, and rare in private practice.

PATHOLOGICAL ANATOMY.—The lesion is peculiarly characteristic; it is a necrobiosis. There is first reddening and swelling of the gum, then separation of the swollen border of the gum from the tooth, then a purulent exudate on the edge of the gum. Beneath this exudate takes place necrosis and softening of the tissue, leaving an ulcer. Loss of tissue substance is characteristic of all lesions produced by the bacillus fusiformis and its associated spirocheta, while a comparatively slight inflammatory manifestation is seen in the tissue adjoining the lesion. The amount of gum destruction is variable; in some cases a large part of the root of the tooth may be denuded.

The process may remain limited to the gums, but usually extends to the mucous membrane of the lips, cheeks, and edges of the tongue. The ulcers resemble those seen on the gums.

SYMPTOMS.—Stomatitis ulcerosa is usually preceded by moderate constitutional symptoms, such as fever, loss of appetite, and fretfulness. The mucous membrane of the gums at the free margin of the teeth becomes reddened and soon begins to swell. The normal curve of the gum becomes almost a straight line and covers the lower part of the teeth. The mucous membrane then begins to change in color and becomes purplish. Extreme congestion and softening of the tissues allow hemorrhage to take place from the slightest pressure. Although the anterior surface of the gums is most commonly affected, yet in severe cases the posterior surface also is involved. As the process develops further, the gum becomes more and more loosened as it extends over the teeth. Finally loss of substance leads to the formation of irregular ulcers with yellowish bases, involving the gums, and often involving the lips, cheeks and edges of the tongue.

The necrotic tissue has a characteristic marked fetid odor, which is conveyed by the breath, and by all the secretions of the mouth.

The lymph nodes of the neck are usually enlarged.

The ulcers begin to heal after a period of about a week. The exudate then comes off, and cicatrization is rapid.

DIAGNOSIS.—The lesion is so peculiar that inspection of the mouth usually suffices in making a diagnosis. The gum may be purple and swollen in scorbutus, but when actual ulceration has occurred, the lesion is characteristic of ulcerative stomatitis. The characteristic fetor of the breath is also of aid.

To render the diagnosis sure, coverglass preparations may be made from the exudate covering the ulcers. These are best stained with carbol-fuchsin, the slide being flooded with the stain, and then immediately washed with water. The large bacilli of variable size, some of them very long, with pointed ends, staining deeply, and the delicate cork-screw shaped spirochetae, staining faintly, are unmistakeable.

PROGNOSIS.—The disease tends to recover spontaneously, but if untreated, it may run a long course, and become a serious drain on the child's vitality. In sickly children, stomatitis ulcerosa may turn the scale against them. With treatment, improvement is very rapid.

TREATMENT.—Proper care of the mouth and teeth is usually efficient prophylaxis against ulcerative stomatitis. When the disease has developed, the local treatment of the mouth, and the management of the feeding, should be carried out in the same way as for catarrhal stomatitis.

In addition to washing out the mouth, the disease is best treated by internal administration of chlorate of potassium. Chlorate of potassium must, however, be given with great precaution to infants and children, as in certain cases it acts as a poison, some infants being affected by even minute doses. The symptoms which show that chlorate of potassium is producing deleterious effects in infants are drowsiness and suppression of urine, with weakness of the heart and sometimes cyanosis. When these symptoms follow the administration of the drug, it should be omitted at once. Chlorate of potassium when given internally has been found to be secreted in the saliva within five or ten minutes, and thus has an opportunity of producing a direct effect upon the lesions of the gums. The doses of chlorate of potassium which it has been found can be safely administered to infants and children, should be remembered when prescribing the drug. The table gives the minimum doses which can safely be taken in the twenty-four hours at different ages, and which are sufficient to produce the specific effect of the drug in treating cases of stomatitis ulcerosa.

TABLE 66

Amount of Chlorate of Potassium Which Can Be Safely Given in Twenty-four Hours at Different Ages

AGE	GRAINS
Under 1 year	10
1 to 2 years	15
2 to 6 years	20
6 to 8 years	25
8 to 14 years	30

In order that the chlorate of potassium shall produce the best effects it should be given frequently. The total amount for twenty-four hours which is to be given at any special age is to be placed in a tumbler and dissolved in as many tablespoonfuls of sterilized water as there are doses to be given within the twenty-four hours. Let us suppose that the number of hours which the child sleeps is ten; then fourteen tablespoonfuls of the solution should be prepared, and the child should be given one tablespoonful every hour when it is awake.

Under this treatment the disease is ordinarily cured in a week or ten days. The treatment should, however, be continued for a number of days after the mouth is apparently entirely well.

When extensive ulceration has taken place, its disappearance may sometimes be expedited by the application of nitrate of silver.

NOMA

(Stomatitis Gangrenosa; Cancrum Oris)

Noma is a comparatively rare disease, characterized by rapidly spreading gangrene, originating in the mucous membrane near a mucocutaneous orifice. The process has been known to originate in the nose, anus, vulva, and prepuce, but as the commonest point of origin is the mouth, the term stomatitis gangrenosa is usually considered as synonymous with noma.

ETIOLOGY.—The preponderance of modern opinion believes that the cause of noma is the same as that of ulcerative stomatitis and Vincent's angina, namely the *bacillus fusiformis* with its associated spirillum, (or spirocheta). The lesions always show a mixed infection, but the etiologic importance of the bacillus fusiformis and the associated spirillum is supported by numerous facts. The great majority of observers have regularly found these organisms in the lesions of noma. The gangrenous character of the lesion of noma bears a close histological resemblance to that of both Vincent's angina and ulcerative stomatitis, and the earliest lesions of noma are indistinguishable from those of Vincent's angina. While other organisms are found in the gangrenous tissues of noma, many observers have reported that only the bacillus fusiformis and spirillum were found in the deeper tissues near the line of demarcation. Some observers have reported peculiar filamentous forms near the line of demarcation, and Tunnicliffe has obtained a similar filamentous growth in cultures of the bacillus fusiformis.

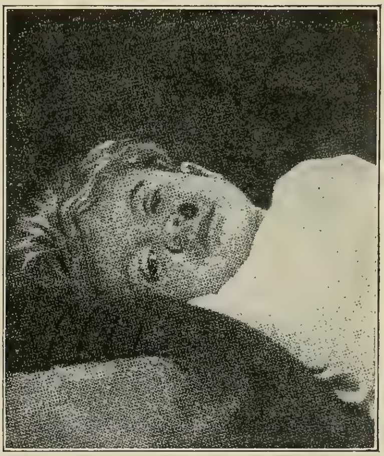
The organisms causing noma are of wide distribution, being often found in apparently healthy mouths. Therefore there must be another etiologic factor in the development of the disease, beside the mere presence of the organisms. This factor is probably low resistance in the individual child. Noma is most common in institutions for children. It is most frequently seen as a sequela of one of the acute infectious diseases, such as measles, scarlet fever, typhoid fever and whooping-cough. It is occasionally seen in children suffering from chronic malnutrition. There is probably a third essential etiologic factor, as noma seldom attacks healthy mouths, but is usually preceded by catarrhal or ulcerative stomatitis. It is possible also that lowered resistance is not the sole factor in determining that the infection shall take the severe form seen in noma, but that variation in the virulence of the particular strain of organism may be the determining factor. This possibility is strengthened by the fact that noma is sometimes seen in small epidemics, and that there is evidence that it can be transmitted by contact.

Noma is particularly a disease of early life, and is most frequent in children under five.

PATHOLOGICAL ANATOMY.—The lesion is a spreading gangrene. There is a certain amount of inflammatory reaction in the newly affected tissues, but suppuration does not occur, and gangrene dominates the picture. In most cases there is little or no tendency toward limitation, and the gangrene continues to spread until death occurs. In some cases a line of demarcation may form and the gangrenous tissue may separate as a slough, leaving a large area to be filled by granulation.

SYMPTOMS.—The first symptom which is apt to be noticed is the gangrenous odor which comes from the mouth. On examination a dusky spot is found on the cheek or lip which soon becomes an ulcer which tends to spread rapidly. The spreading ulcer shows a greenish-black necrotic mass. The cheek becomes much swollen, is hard and edematous, the edema especially affecting the tissues under the eye. The gangrenous process extends very rapidly, at times destroying large portions of the face, and also involving the bones, which become denuded. The teeth become loose and fall out. The odor from the gangrenous tissue is excessive. The flow of saliva is very much increased. The degree of suffering which the children undergo varies very much; sometimes it seems as if they suffered no pain whatever. The temperature varies, at times being raised and again being subnormal. The pulse is weak and rapid. The appetite is diminished, and the children are likely to have diarrhea, probably due to the infectious nature of the products of the mouth which are swallowed. Hemorrhages are rather rare, as the blood-vessels are usually filled with thrombi. Secondary affections, such as catarrhal pneumonia from the inhalation of septic material, are not uncommon. The child may die from one of these

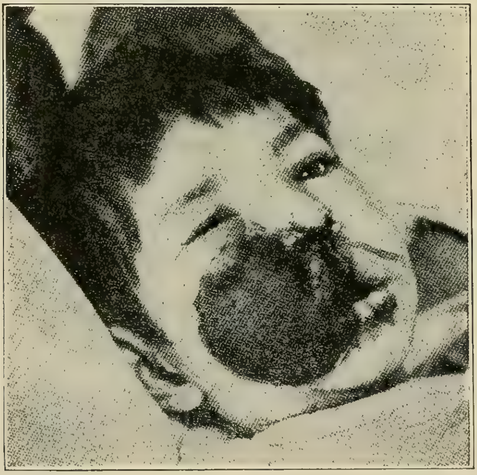
FIG. 227



Stomatitis gangrenosa, left cheek. Female, 4 years old secondary affections, or it may become more and more weakened by the local condition, and unless the morbid process is arrested it will die eventually from exhaustion. The average duration of the disease is one week.

DIAGNOSIS.—The diagnosis of this disease, except in its earlier stages, is not difficult. At times, however, a local ulcerative process produced by a decayed tooth may simulate closely stomatitis gangrenosa. In these cases the diagnosis is made more difficult by the fact that the tissues of the cheek may become hard and look as though perforation might take place. Coincidently with this condition the ulceration of the gum and often of the mucous membrane of the cheek, with the foul odor which emanates from it, makes the similarity of the two diseases very striking. In simple ulceration from a tooth, however, active local treatment with solutions of myrrh or of soda combined with frequent washing of the mouth with sterilized water is soon followed by marked improvement, while when stomatitis gangrenosa is present the morbid process continues to extend.

FIG. 228



Stomatitis gangrenosa secondary to measles and pneumonia. Female, 5 years old

PROGNOSIS.—The prognosis in cases of stomatitis gangrenosa which are untreated is almost universally fatal. Cases have been known, however, in which a line of demarcation has formed around the gangrenous spot, granulations have arisen, and cicatrization has followed, leaving extensive scars. If this disease is treated by extirpation of the diseased tissues in the very beginning, the prognosis becomes more favorable. When the disease has perforated the cheek and the gangrenous process has become extensive, the child is seldom relieved even by surgical treatment.

TREATMENT.—Care should be taken when a child is affected with a disease of an exhausting nature that its mouth be kept thoroughly cleansed, for we can never tell when or in what individual the mucous membrane may become vulnerable to the organism which produces stomatitis gangrenosa. In noma it is very important for the success of the treatment that it should be begun very early in the disease. When the diagnosis has been definitely made, it is wiser not to temporize with applications of nitrate of silver and other drugs, but at once to place the case in the hands of a surgeon and have the entire area of the invaded tissues excised. It is also well after the gangrenous process has become removed by the knife to destroy an area of healthy tissue by means of cauterization. There should be no delay in operating upon these cases, as great destruction of the tissues may take place in even a few hours.

After the operation the tissues should be inspected frequently, to see whether there is any return of the gangrenous spots, and, if found, these spots should be removed immediately. As the disease is very apt to recur, plastic operations to obviate deformity should not be undertaken very early after the operation.

PROBLEMS AND RESEARCH.—The principal problems to be investigated in noma are connected with the microörganisms causing the disease. The relative parts played by the bacillus fusiformis with its associated spirillum, and by other organisms, still offer a field for investigation. Most of the problems in noma are connected with the bacillus fusiformis, its relation to the spirillum, and its biology in general. This will be more fully considered under the heading of Vincent's Angina.

BEDNAR'S APHTHAE

(Ulcera pterygoidea). (Ulceration of the Palatine Angles)

This is a disease, first described by Bednar, seen only in the newborn, or in infants in the early weeks of life, and characterized by ulceration of the mucous membrane of the hard palate. The ulcers are usually bilateral and symmetrical, and are situated near the hamuli pterygoidei. They may be joined by a bridge, or extend, giving various configurations. Ordinarily they are about the size of a pea, are superficial, covered with a yellowish-white mass, and surrounded by a red areola. They are painful, interfering with sucking.

The lesions in this particular situation are probably caused by the fact that the mucous membrane of the mouth is most tightly stretched over the hamular process of the palate, and that trauma, such as too vigorous mouth washing, or the prolonged sucking on the rubber nipples, produces injury to the mucous membrane.

In prophylaxis, forcible washing of the mouth, and the use of rubber nipples and pacifiers, must be avoided. If a two per cent solution of nitrate of silver is daily applied to the ulcers, they heal very rapidly.

RANULA

Beneath the tongue we sometimes find the mucous membrane bulging out as a bluish, translucent tumor which is soft, painless and semi-fluctuating. This condition is called *ranula*, and is a retention cyst caused by the blocking of a mucous duct. When opened, a small amount of glairy fluid escapes, but the collapse of the walls of the cyst brings the edges of the cut together and they quickly adhere. The fluid will soon re-collect; therefore the only sure way of dealing with these cysts is to pinch up their anterior wall with fine forceps, and with the scissors remove so much of it as to leave no opportunity for the edges to adhere. A gentle application of nitrate of silver to the edges and interior of the sac after the cut has been made with the scissors materially helps to promote the cure.

FISSURES OF THE LIPS

Cracks and fissures of the lips are common in children. They occur most commonly in cold weather. The vermillion border of the lip becomes dry and rough, and the discomfort causes the child to moisten the lips with the tongue, which increases the condition, so that eventually one or more deep, painful fissures are formed.

The lips should be kept covered as much as possible with some simple ointment. Deep fissures should be touched with the nitrate of silver stick.

PERLÈCHE

(Epstein's " Faule Ecken") (" Bad Corners," " Foul Angles " of the Mouth)

This is a disease, first described by Lemaistre in 1886, and exhaustively described by Epstein in 1900, characterized by ulceration at the corners of the mouth.

ETIOLOGY.—There is evidence in support of an infectious cause for this condition. It is seen mainly among school-children, and family epidemics have been reported. No particular microörganism has as yet been associated with the lesion.

SYMPTOMS.—The disease is limited to the angles of the mouth. Radiating fissures are formed, which cause pain and burning leading the children to lick the affected parts, and to suck air to the corners of the mouth. The constant licking and irritation leads to painful ulceration, the ulcers being covered with a grayish exudate.

DIAGNOSIS.—The ulcers are not unlike the mucous patches of hereditary syphilis, but are distinguished by their strict localization, by their more superficial character, and by the fact that they are more painful. The history and absence of other syphilitic lesions are important in the diagnosis of doubtful cases.

PROGNOSIS.—The majority of cases will heal spontaneously even without treatment. The disease is more protracted if proper treatment is not employed. Occasionally, even with treatment, the disease may be prolonged for weeks or months. Healing occurs without scar formation.

TREATMENT.—The crusts covering the ulcers should be softened with boracic acid ointment, and removed. The exposed ulcers should be touched with silver nitrate daily, and kept covered with boracic acid ointment.

HERPES LABIALIS

("Cold Sore")

This is very commonly seen in the acute febrile diseases in children, but sometimes occurs alone. A group of vesicles form on the lip, which rupture, with the formation of crusts. Healing occurs spontaneously, but is retarded by the tendency of children to pick at the crusts. Picking at the lips should be prevented, and young children should wear elbow splints at night. The crusts should be softened by an application of boracic acid ointment. After softening, the ointment should not be used further, but the lesions should be washed daily with a boric acid solution, and dusted with boric acid powder.

GLOSSITIS

Glossitis is so rare a disease in children that the possibility of its occurrence only need be mentioned. In this affection there is an acute inflammation of the tissues of the tongue, accompanied by fever, enlargement of the organ, and considerable pain. There is usually a hypersecretion of saliva, and at times the obstruction to respiration from the occlusion of the throat by the greatly enlarged tongue produces somewhat alarming symptoms, though, as a rule, not serious ones.

This disease may be caused by direct injury to the tongue from corrosive substances, by heat, or by the stings of insects, and sometimes probably by sepsis. It runs a variable course; it is not especially serious, and tends to recover after a few days. The treatment is purely symptomatic. The frequent local application of ice-cold alkaline solutions to the tongue and mouth is indicated.

LINGUA GEOGRAPHICA

(Geographical Tongue) (Mappy Tongue)

(Annulus Migrans) (Glossitis Areata Exfoliativa)

ETIOLOGY.—This condition appears most often in early childhood. The cause is unknown. The most probable explanation of the condition is that it is a manifestation of some constitutional anomaly. Czerny believes that it may be one manifestation of his "exudative diathesis" as he saw it frequently in children of that type.

SYMPTOMS.—Grayish-white areas which rapidly enlarge appear upon the surface of the tongue. The centers of the areas change to a red discolorization, while the grayish-white borders extend in crescentic outline. Thus irregular figures are formed, and when several such figures coalesce, the characteristic map-like appearance of the tongue is seen. The reddish discolorization eventually fades to the normal, beginning in the center, but in the meantime new areas may appear.

The disease is a very benign one, causing no discomfort or functional disturbance, and never leading to ulceration, nor to any serious complication. It runs a very chronic course, and may recur periodically for months or years. The changes in the tissues are confined to the upper layers of the epithelium, the advancing borders representing circumscribed epithelial hyperplasia, while the reddened areas represent epithelial exfoliation.

The only importance of the disease is the alarm which it causes when discovered by the child's mother. No treatment is of any benefit.

ULCER OF THE FRENUM

The frenum of the tongue is sometimes the seat of ulceration in infants. The process may erode more or less of the frenum, and may even extend into the tissues of the tongue. The ulcers are covered with whitish exudate, and are surrounded by an elevated border.

The ulcers are caused by pressure of the frenum against the sharp edges of the lower central incisors. It is most commonly seen in whooping-cough, where the tongue is pressed and rubbed against the teeth during the paroxysm. Except in whooping-cough it is only seen in infants who have the two lower central incisors only. In such cases the ulceration is caused by friction during sucking.

The ulcers heal readily when touched with alum or nitrate of silver. The teeth may be covered during feeding with a little absorbent cotton.

DIFFICULT DENTITION

The normal development of the teeth in infancy is a physiological process, and in the majority of instances it is attended by no morbid symptoms of any kind. On the other hand various disturbances are frequently seen during the teething period. Two views have been held as to the significance of these symptoms. According to the older view, a great variety of diseases and pathological symptoms occurring during the teething period were attributed to this cause, and dentition was supposed to be an important etiological factor in a large number of diseases occurring between the ages of six months and two years. According to a modern view held by some observers, dentition never causes either subjective discomfort or reflex symptoms, and all diseases occurring during the teething period should be looked upon merely as coincident.

Both of these views are extreme, and neither represents the facts. The list of diseases formerly believed to be connected with dentition has been gradually shortened, as our increasing knowledge has shown them to depend wholly upon other causes. Thus we no longer regard such conditions as cough, diseases of the respiratory system, and diseases of the skin (except possibly eczema), as in any way connected with teething. The prevalence of this old view has done much harm, in leading physicians to overlook many serious diseased conditions, while attributing the symptoms to teething. Nevertheless, although the importance of dentition as an etiological factor has been greatly exaggerated, the modern view can in no way be made to agree with the experience of the majority of careful observers. There is no doubt that many symptoms, sometimes even of a serious and alarming nature, can be due to no other cause than the eruption of the teeth. The definite appearance of these symptoms with the cutting of a tooth, their relief when the tooth has come fully through the gum, and their recurrence with the cutting of a subsequent tooth, leave no doubt as to their cause.

SYMPTOMS.—The symptoms of difficult dentition vary greatly in severity. In healthy children, at least half have no symptoms of any kind. In children who do show disturbance, the symptoms are usually comparatively mild except in children who are delicate or rachitic, or with highly-organized nervous systems.

The most common symptom is loss of appetite, young infants refusing to take the breast or bottle, and older infants refusing solid food. Teething babies will often begin to suck eagerly as if hungry, and then stop in evident discomfort; in other cases they do not seem hungry. The amount of food taken may be so little that their nutrition is seriously interfered with. In addition to loss of appetite there is frequently seen a languid irritability and fretfulness, with crying during the day, and at night restlessness, wakefulness, or a restless sleep. During the existence of these symptoms the infant's weight may be stationary, or there may even be loss of weight.

The connection of these symptoms with the cutting of the teeth is often shown by signs of local irritation in the mouth. There is often much evident discomfort in the region of the gums, the infant continually rubbing them with its fingers, and seeming to get relief from biting on hard substances. The mouth frequently becomes hot, and sometimes dry, although hypersecretion of saliva with profuse drooling is more common. In some cases the mucous membrane of the mouth shows the condition of catarrhal stomatitis.

There are certain other disturbances which can only be explained through reflex nervous disturbance. Fever is not uncommon during the cutting of a tooth. In mild cases in healthy children the temperature is usually only moderately elevated, to 100° or 101.5°. In delicate children, and occasionally in healthy children, the temperature may rise to 102°, 103°, or even to 104°. I have seen several apparently healthy children, who have shown, with the cutting of each tooth, a marked febrile reaction. Congestion of the drum membrane of the ear is common during the teething period, and this may go on to the development of a real otitis media. This congestion of the ear has been explained on a basis of nervous connection through the otic ganglion, with which the inferior dental nerve connects. While it is possible that dentition is an etiologic factor in congestion of the ear, it is probable that the connection is no more than a coincidence.

Digestive disturbances occur in teething children with such frequency, that we cannot but conclude that there may be some etiological connection. Such disturbance can be explained only on the basis of reflex nervous disturbance which influences the alimentary secretions, thus lowering digestive power. The most common manifestation of lowered digestive power associated with teething is the appearance of undigested or green movements. In some instances diarrhea or vomiting recurs in definite association with the cutting of a tooth.

Convulsions have been observed with no other apparent cause than dentition. That the reflex disturbance from dentition may be severe enough to cause convulsions is possible, but instances are quite rare.

There are two stages in the course of dentition when symptoms appear. One is an earlier stage when the growth of the tooth in its socket is first becoming active, but the tooth is not yet pressing on the gum. The other stage is when the tooth is actually pressing upon and forcing its way through the gum. The loss of appetite, general irritability, failure to gain in weight, and tendency toward digestive disturbances are symptoms common to both stages. Fever also is seen in either stage, but is more common in the earlier, before the tooth is coming through the gum. The local symptoms pointing toward the mouth are seen only in the later stage. The stages should be remembered, as it is not necessary to find a swollen gum, or any signs of local irritation in the mouth, in order to make a diagnosis of difficult dentition.

DIAGNOSIS.—*The diagnosis of difficult dentition should never be made except through the most rigid and careful exclusion of all other possible causes for the symptoms.* The mere fact that a baby is teething should never warrant the assumption that such symptoms as languid irritability, loss of appetite, fever, or digestive disturbance are due to the cutting of the teeth. The child should be observed most carefully, and frequent thorough physical examinations should be made. In many cases it will be possible to trace the symptoms to some other cause. The ears should be examined with especial care. I have often seen cases with fever diagnosed as difficult dentition, in which the examination of the urine had been neglected, but which finally were shown to be cases of pyelitis. Only when the symptoms in a teething baby disappear with the coming through of the tooth, is the correctness of the diagnosis assured.

TREATMENT.—Feeding should never be forced in cases of difficult dentition with loss of appetite. The diminution in the appetite is not wholly due to the discomfort in the mouth, but is often a manifestation of nature's effort to protect the lowered digestive power of the infant against overload. Even if the infant is actually undergoing loss of weight from insufficient food, starvation will never become dangerous when caused by teething, and there is greater danger of producing digestive disturbance from relative overfeeding. In teething babies who show other signs of disturbance, but who are taking plenty of food, it is often well to dilute their milk, and to reduce the quantity given, and if they actually show digestive disturbance, these measures are imperative.

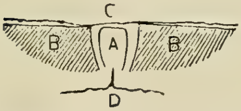
A simple measure which is often very effective in teething babies, is to squirt a few drops of cold water from a medicine dropper over the gums immediately before each nursing. This not only causes the baby to take more food, but appears to add greatly to its comfort. This measure may be repeated between feedings, if it appears to give relief.

Bromide of sodium is often of great value in difficult dentition, and is indeed the only drug which should be employed in this condition. It may be given to infants from six months to two years of age in two-grain doses, either three times daily, or every four hours, according to the severity of the symptoms and the degree of relief attained.

The question of lancing the gums in cases of difficult dentition is one which has given rise to much discussion, and to very diverse opinions. Whether or not this procedure is indicated depends on the stage of dentition in which the symptoms appear.

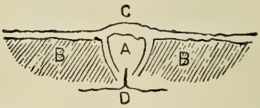
Figures 229 and 230 represent the condition of the gums in relation to the teeth in the two stages described above. Figure 229 represents the mucous membrane over the crown of the tooth as flat and on a level with the rest of the gum. This is the condition of the gum in the majority of cases of difficult dentition, yet very severe symptoms may arise in these cases. In this condition the gum should never be lanced. In figure 230 the mucous membrane covering the crown of the tooth is shown to be markedly raised above the level of the gum. In these cases, symptoms of local origin and often of great severity arise. The infant evidently has extreme pain and tenderness in its mouth. It cries incessantly, and often refuses to take its nourishment, on account of the acute pain which it suffers, and also of the tenderness which is produced by the least pressure on the gum, so that it may become weak and exhausted. There is usually a considerable elevation of the temperature to 102°, 103°, and 104°.

FIG. 229



A, tooth in bone socket; B, jaw-bone; C, gum, soft, not inflamed or swollen; D, dental nerve.

FIG. 230



A, tooth in bone socket; B, jaw-bone; C, gum, tense, inflamed, swollen; D, dental nerve.

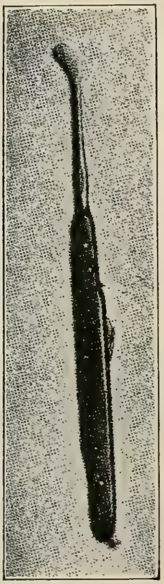
Vomiting is not uncommon, and there is twitching to such an extent that convulsions seem to be threatening, and at times actually occur. There are also great restlessness and insomnia.

In these cases lancing the gum affords immediate relief. The temperature quickly subsides, the pain and general nervous symptoms disappear, and the infant after sleeping quietly for an hour or so wakes up and takes its food with avidity. The treatment in this class of cases, when the diagnosis is once made, is to lance the gum. This is done in the following way. The infant is placed in the nurse's lap, with its head in the lap of the physician, the nurse holding its arms firmly. The physician, after having first thoroughly sterilized his hands and washed the infant's mouth and gums with sterilized water, carefully makes an incision over the swollen gum well down to the crown of the tooth. Figure 231 represents the gum-lancet which I am in the habit of using for this purpose. As only the end of this lancet is sharp, there is less danger of wounding the infant's lips and mouth than with the ordinary bistoury. Before using the lancet it should be thoroughly sterilized.

Although much has been said about the danger of hemorrhage in these cases, and of infection of the wound by pathogenic organisms, yet instances in which such results have occurred are so exceedingly rare that they should not deter us from treating the case properly as we would treat an abscess in the mouth, tonsil, or pharynx. It has also been said that a cicatrix may form on the gum over the crown of the tooth as a result of lancing. This is an exceedingly rare occurrence, and need scarcely be taken into account. The probability is that when such an instance has occurred, the case was not one in which the gums should have been lanced, and the fear of such a result as this should certainly not weigh in the balance against the possible exhaustion and acute pain which may continue for days unless relief is given by cutting.

It must be understood, however, that the first class of cases of difficult dentition are by far the most frequent.

FIG. 231



Gum-lancet

II. DISEASES OF THE NOSE

ACUTE RHINITIS

(Acute Coryza.) (Acute Naso-Pharyngeal Catarrh.)
(Common Cold.)

This is a catarrhal inflammation, which starts most commonly in the naso-pharynx, which involves the mucous membrane of the nose, and which may extend to the tonsils, pharynx, larynx, trachea and bronchi, and to the accessory sinuses and middle ear. It is called Rhinitis or Coryza because the symptoms are chiefly nasal at the start. The disease is particularly common both in infancy and childhood.

The terms " coryza," " rhinitis," and " cold " are used to describe certain symptoms, without reference to etiology, and as commonly employed include not only true inflammation, but also various disturbances of function, secretion, or sensation which cannot be classified under any more definite pathological heading.

ETIOLOGY.—The etiology of the common cold has been, and still is, the subject of much discussion, and of very diverse opinions. Various factors have been etiologically connected with this symptom-complex, and these may be divided into predisposing causes and exciting causes. The following have been associated with acute rhinitis as predisposing causes:—

Hereditary Constitutional Predisposition.—Individual children are occasionally seen who are particularly subject to repeated colds, in whom the tendency to take cold cannot be explained on any other basis. In other words, in these children there is neither faulty hygienic conditions, nor undue exposure to cold, irritants, or contagion.

Acquired Predisposition.—Colds are particularly common in children who are subjected to certain errors in hygienic management. They do not get enough fresh air, as they are kept in-doors much of the time, and are not allowed to go out unless the weather is particularly fine, for fear that they may " catch cold." They live in overheated rooms, and sleep in rooms with the windows closed. The fear of their taking cold leads to their being so over-clothed that they cannot engage in any exercise without a profuse perspiration.

This etiologic factor has no bearing on the question of the actual exciting causes of colds. These faults of hygiene cause the nasal mucous membrane to be more sensitive, and such increased sensitiveness can act as a predisposing cause, whether the exciting cause be irritants from without, or exposure to cold, or infection with pathogenic microörganisms.

CONSTITUTIONAL DISEASE.—There is evidence based on the frequency of colds in poorly nourished, or rachitic children, that these conditions may predispose toward colds.

LOCAL DISEASE.—Children with adenoids, or with subacute or chronic tonsillitis, are particularly liable to colds.

The following have been associated with acute rhinitis as *exciting causes.*

EXTERNAL IRRITANTS.—Mechanical, chemical, or thermal irritation will cause functional disturbance of the naso-pharyngeal mucous membrane in certain cases. Examples of this kind of exciting causes are trauma, the breathing of formaldehyde gas, the breathing of dry overheated air, and the so-called " rose cold," in which the irritation comes from the pollen of a plant.

EXPOSURE TO COLD.—In certain susceptible individuals, rhinitis appears to follow directly some form of exposure to cold. Sitting in a draught, exposure to damp cold without proper clothing, " cooling off " rapidly after profuse perspiration, falling into water, or otherwise becoming chilled, throwing off the bed-clothes at night—all these have appeared to act as immediately exciting causes. Much of the recent discussion has had to do with the relative etiological importance of exposure and infection.

INFECTION WITH MICROÖRGANISMS.—The clinical course pursued by the most typical common colds, those which form the majority of the cases met with in practice, is of such a character, that there can be no doubt that they represent *bacterial infections.* Further evidence is derived from the fact that coryza is a prominent symptom of several infections of which the exciting microörganism is known, such as influenza and measles. Finally, continually accumulating evidence has firmly established the *contagiousness* of most typical colds.

What proportions of colds are due to bacterial infection? Many observers state that all colds are of infectious origin. This is an extreme statement, when considered under the very wide definition of the terms cold, acute rhinitis, acute coryza, as commonly employed. We know that " hay fever," " rose colds," are not due to bacteria. Leaving aside these instances, *the prevalent view is that the majority of common colds are bacterial infections.*

THE MICROÖRGANISMS CAUSING RHINITIS.—The prevalent view is that acute rhinitis is caused by a great variety of microörganisms, among them being numbered the staphylococcus, pneumococcus, streptococcus, micrococcus catarrhalis, and the influenza bacillus. Any of these organisms may be present in the naso-pharynx, but the mucous membrane is normally resistant to actual infection. Some condition lowering the resistance of the mucous membrane is required for infection to take place. The various predisposing causes produce a chronically lowered resistance, explaining the frequency of colds in certain individuals. External irritation and exposure to cold, while apparently exciting causes, act by producing a vaso-motor disturbance which lowers the resistance of the mucous membrane and allows infection to occur. Colds may also occur, not by autoinfection, but by contagion.

The above is the view most generally held as to the etiology of acute rhinitis. In my opinion it is not wholly satisfactory. I believe it tends too much to exaggerate the importance of exposure to cold and of autoinfection, and to minimize the importance of contagion. The evidence in favor of *direct transmission* being the usual method of infection in common colds, is continually piling up. The evidence in favor of exposure being the exciting cause of infectious colds is constantly appearing to be less, and in my experience is very little. I believe that *nearly all, if not all, infectious colds are excited not by exposure to cold, but by contagion.*

The evidence in favor of contagion throws grave doubt upon the generally accepted bacteriology of acute rhinitis, and raises the question whether the cause of the common infectious cold is not *some specific microörganism as yet undiscovered.*

CLASSIFICATION.—I would suggest the following provisional classification for acute rhinitis.

1. Vaso-motor colds—
   a. From external irritants.
   b. From exposure to cold.
2. Infectious colds—
   a. Symptomatic of known specific infections.
   b. Non-contagious—probable autoinfection.
   c. Contagious—probable specific organism.

Vaso-motor colds from external irritants include "rose colds." In other conditions where an external irritant plays the exciting rôle, the symptoms are much like those of hay fever, and I do not believe infection plays any important part.

Vaso-motor colds from exposure include those cases in which the evidence is plain that exposure to cold is immediately followed by symptoms of rhinitis. Usually in such cases the symptoms occur repeatedly. In my experience the cases of this kind differ greatly clinically from the common infectious cold. These children suffer from repeated colds following the slightest exposure, but the disease does not follow the typical course of the common infectious cold. There is less swelling of the mucosa, the discharge remains serous throughout, there is little if any fever, and the process does not extend to the ear, tonsils, larynx, or accessory sinuses. I do not believe that this condition represents an infection, or that it is often the starting point of an infection, but that it is purely a vaso-motor disturbance. It is most commonly seen in children who have acquired a predisposition through faulty hygiene, as described above, but is rare in comparison with the infectious cold.

The symptomatic coryzas, such as are seen in measles, require no explanation.

The non-contagious infectious cold is seen mainly in adults, being very rare in children. It is symptomatically much like the common contagious cold, but differs enough to make it probable that it is due to some other organism. The cases generally run a more prolonged course, and the secretion is often purulent from the start.

The common contagious cold is the type which will be described in detail.

SYMPTOMS.—The disease usually begins suddenly with a sense of fullness, burning, and dryness in the nostrils, with sneezing, and with difficulty in breathing through the nose. The entrance of air into the naso-pharynx is blocked by the swelling of the erectile tissues covering the turbinate bones, and almost complete occlusion takes place. In the course of a few hours appears a serous discharge, which later becomes mucopurulent; in severe cases in infants there may be a very profuse purulent discharge. There is usually a slight rise of temperature, but in infants the temperature may be high, reaching 104° or 105°, while in older children the temperature may remain normal throughout. Although the general symptoms are often slight, there is commonly a very evident sense of discomfort, with loss of appetite and general malaise. The nasal obstruction in infants produces difficulty in nursing, which is frequently interrupted. The patient is often forced to breathe with the mouth open, and a resulting condition of dryness of the mucous membrane of the mouth and throat causes a choking sensation. Sleep is often much disturbed. The natural tendency of an infant or young child is to keep the mouth shut, so that often when the nose is occluded it breathes with great difficulty when asleep, and its face becomes congested and even cyanotic. In severe cases there may be loss of the senses of smell and taste.

Extension of the process through the lachrymal ducts may give rise to a conjunctivitis. Extension of the process to the pharynx or tonsils may cause pain on swallowing. Extension of the process to the larynx and trachea may cause cough and hoarseness. Closure of the Eustachian tubes may cause deafness or earache. The most common complication is otitis media, which should be constantly watched for. Other complications occasionally seen, especially in severe cases, are cervical adenitis, bronchitis, bronchopneumonia, and retropharyngeal or peritonsillar abscess.

DIAGNOSIS.—Other conditions which give rise to symptoms of nasal catarrh in children are measles, influenza, nasal diphtheria and hereditary syphilis. In measles and influenza the fever and constitutional symptoms are more severe in proportion to the nasal manifestations than in the common infectious cold. In nasal diphtheria there is often a profuse discharge which is frequently blood-tinged. Whenever a blood-tinged discharge is present, or whenever the nasal discharge persists longer than a week, the possibility of diphtheria should be suspected and cultures should be taken. Syphilis also is suggested by the persistence of a nasal discharge, and the diagnosis can only be confirmed by the appearance of other syphilitic manifestations.

PROGNOSIS.—In healthy children the prognosis of these infectious colds is usually good. The disease ordinarily runs its course in a variable period of from three days to a week, although severe cases may last longer, and unless the child is subjected to fresh exposure it recovers entirely. In spite of the favorable prognosis in healthy children, these infectious colds are far from being such trivial affections as is generally supposed. They exercise a markedly unfavorable influence upon nutrition, and in cases where nutrition is already difficult, or in debilitated children, the occurrence of a cold may prove quite serious. The complications of acute rhinitis are also more frequent and more serious in poorly nourished infants. I have seen puny, ill-cared for infants die from a simple acute rhinitis with occlusion of the nares.

TREATMENT.—Young children with acute rhinitis should be kept in-doors, in a well aired room, at an even temperature of about 68° F., during the acute stage. If there is fever, they should be kept in bed. One or two teaspoonfuls of castor oil should be given to infants at the beginning of the attack; this is not necessary in older children, but the bowels should be kept open. It is well to reduce the quantity of food, especially in infants. At times, however, feeble infants will not take enough milk, owing to the difficulty of sucking produced by the nasal obstruction. In such cases, milk should be given with the spoon.

I believe quinin to be useful in infectious colds; from one-half to two grains may be given three times a day, according to the age of the child. Quinin appears to increase resistance, and is particularly useful in prolonged cases. Tincture of euphrasia, in doses of ten drops every hour for four or five doses each day, will often lessen the nasal secretion, and is good routine treatment for infants. If this fails, the only drug which will lessen the nasal secretion is belladonna. The following prescription is useful:

℞	1-6 mos.	6-12 mos.	1-2 yrs.	2-6 yrs.
Tincturae Belladonnae..........	m iv	m vi	m viii	m x
Pulveris Camphorae.............	gr. ii	gr. ii ss	gr. iii	gr. iii
Sacchari Lactis..................	q.s.			

M. Div. et ft. tabellae no. xxx.
Sig. One tablet in water every two hours.

For local use, liquid albolene may be instilled into the nostrils. The following preparation often gives great relief:—

℞	
Menthol..	gr. 1/2
Oil of Eucalyptus................................	m x
Liquid Albolene..................................	℥ i

M. Sig. 3 drops in each nostril every two hours. In older children, this may be advantageously given with the oil atomizer.

Irrigation of the nose with mild alkaline antiseptic solutions, such as Seiler's solution, is often more valuable than the oily spray, but it should be very carefully given, without force, on account of the danger of carrying the infection into the Eustachian tubes. I believe it is better to avoid irrigation except in severe cases.

When there is so much nasal obstruction that respiration or sucking is seriously interfered with, a few drops of adrenalin solution (1 to 1000) diluted with saline solution in the proportion of one drachm to the ounce, may be instilled with a medicine dropper. The excoriation of the upper lip from the discharge should be prevented by the application of vaseline.

PROPHYLAXIS.—I believe the most important measure in prophylaxis against acute rhinitis is the prevention of contagion. Whenever any member of the household is suffering from an infectious cold, the infant or young child should be guarded against contagion with the same rigidity as it would be against scarlet fever or diphtheria. Isolation and quarantine of the affected adult member of the family not being practical, the uninfected child should be rigidly isolated and quarantined in every household where it can have its own room and attendant. Young children should never be taken to moving-picture shows, into large department stores or other crowded places, or into the street cars if it can possibly be avoided. Other frequent spreaders of contagion are kindergartens, schools, dancing-schools, churches, Sunday-schools, and children's parties. Children who are subject to colds should be kept away from all of these.

A second important measure in prophylaxis relates to the hygienic surroundings of the child. The faulty hygienic surroundings described under the head of Etiology as leading to acquired predisposition, should be corrected. Children should be accustomed to going out doors daily, unless the weather is especially bad, should live in rooms never above 68°, and should sleep in well ventilated rooms. They should not be overdressed. When the predisposition to colds is already acquired, it is difficult to correct the conditions, as the child has already become sensitive to exposure. Such children should be accustomed to live in the open air as much as possible in summer, and to sleep at night in rooms with well-opened windows. Begun in this way, the practice can be carried through the autumn into the winter. The habit of coddling delicate children is however, difficult to correct.

These children should be sponged with cold water, particularly about the chest, throat and spine, immediately upon rising. They should not be allowed to wear chest protectors of any kind. Their underclothing should be woolen both in winter and summer, but should be of medium weight in winter, and of the very lightest weight in summer.

Adenoids should be removed when present in children subject to repeated colds.

Cod liver oil and iron will never eradicate the tendency to colds while hygienic surroundings are not corrected.

PROBLEMS AND RESEARCH.—The chief field for research in acute rhinitis is its bacteriology. The main question to be decided is whether the common infectious cold is an autoinfection with a variety of microörganisms, or whether it is a specific infection. The increasing evidence in favor of contagion as the most common mode of infection, is strongly suggestive of the latter supposition.

Tunnicliffe has discovered in acute rhinitis a delicate, curved bacillus which is as a rule not found in the normal nose. In many cases of acute rhinitis a practically pure culture of this organism was obtained by anaerobic methods. With cultures of this organism were produced not only experimental rhinitis with the recovery of the organism in pure culture, but also specific opsonin and complement-binding antibodies. The finder has called this organism the *Bacillus rhinitis*.

Kruse obtained a filterable virus in the secretions of acute rhinitis, which produced rhinitis when inoculated into the noses of a series of individuals. He reported his filtrate sterile, and consequently regarded the result of his experiment as due to the action of an ultra-microscopic organism. It is not clear, however, whether in making cultures from his filtrate, he used modern anaerobic methods, and it is possible that the bacillus rhinitis of Tunnicliffe might have been present in his filtrate.

This work of Tunnicliffe and Kruse is now awaiting investigation. In the domain of treatment, a number of observers have reported good results from the use of *hexamethylenamin* in common colds.

CHRONIC RHINITIS

(Chronic Nasal Catarrh.)

The terms chronic rhinitis and chronic nasal catarrh do not represent any definite pathological entity, but are loosely used to apply to those cases in which a chronic nasal discharge is the principal symptom. The discharge may be serous, muco-purulent, or purulent according to the etiology, and may cause swelling and excoriation of the nasal entrance and upper lip.

The causes of this condition are very various. In the first group belong cases which are not due to infection, but to some pathological condition in the nose or naso-pharynx which acts as a constant irritant. The following are the principal conditions in this group.

ADENOIDS.—This condition is a hypertrophy of the lymphoid tissue of the naso-pharynx. It is the commonest cause of chronic nasal discharge in infants and young children, and is more fully described elsewhere. Adenoids are recognized by digital exploration of the naso-pharynx. The treatment of this form of chronic nasal catarrh is removal of the cause.

HYPERTROPHY OF THE NASAL MUCOUS MEMBRANE.—This condition is comparatively rare in infancy and childhood. The hypertrophy usually involves the turbinate bones. Sometimes the mucous membrane becomes hypertrophied in the condition of deviation of the nasal septum. The cause of the nasal catarrh is recognized by examination of the nose. The treatment is operative, and should be carried out by a specialist.

MUCOUS POLYPUS.—This is a pedunculated connective-tissue growth originating from the mucous membrane of the middle turbinate bone. It is rare in children. It does not grow on a healthy mucous membrane, and is always preceded by some morbid condition of the nose. It is often multiple.

The symptoms begin with a nasal discharge followed by nasal occlusion. The diagnosis is easily made with a mirror and a probe. The treatment is the removal of the growth.

FOREIGN BODY.—A foreign body in the nose produces a persistent nasal discharge. A unilateral nasal discharge in a child should always suggest a foreign body in the nostril. Young children not infrequently put into the nose such things as peas, beans and shoe buttons. The treatment of this form of discharge is removal of the foreign body.

The second group of causes of chronic nasal discharge includes various infections. The following are the principal conditions in this group.

INFECTION WITH THE PYOGENIC BACTERIA.—This condition may follow an acute rhinitis, or may develop gradually from the start. It is seen most commonly as a sequel of one of the acute specific infections. The discharge is usually muco-purulent, or purulent, and is often profuse. It often flows backward, causing constant desire to clear the throat, choking, swallowing, and cough. The difficulty in breathing through the nose is noticeable in infants, particularly at night.

The treatment of this condition is with oily sprays. The preparation of menthol and oil of eucalyptus in liquid albolene, recommended for acute rhinitis, is useful here. Irrigation with Seiler's solution should be employed if the discharge is very profuse.

INFECTION OF THE SINUSES.—A persistent nasal discharge without other symptoms may be due to a mild sinus infection. It is recognized when the sinuses are examined by means of transillumination.

NASAL DIPHTHERIA.—Chronic infection of the nasal cavities with the Klebs-Löffler bacillus is by no means rare in young children. Such cases usually show no other symptoms than the nasal discharge. A diphtheria culture should be taken from every case of chronic nasal discharge which cannot at the first examination be definitely attributed to some other cause. These cases are resistant to local treatment, but are very rapidly relieved by the administration of 1500 to 2000 units of diphtheria antitoxin. In spite of the cessation of the discharge, diphtheria bacilli may persist in the nasal cavities, as shown by the cultures. The detailed treatment of these cases is described under Diphtheria.

SYPHILIS.—Rhinitis is seen in hereditary syphilis, and is one of its earliest and most constant symptoms. Snuffles, or a nasal discharge in the first two months of life is always suspicious, and should lead the physician to watch carefully for other syphilitic manifestations. Rhinitis is also seen as a different pathologic condition in late hereditary syphilis. The detailed description of the pathology and treatment of these forms of rhinitis is considered under syphilis.

A third group of causes of chronic rhinitis include certain constitutional factors which are not clearly understood. The following are the principal conditions in this group.

MALNUTRITION.—In certain poorly nourished and weak babies is seen a thin, watery nasal discharge. No other cause than the malnutrition can be found for the symptom in these cases. The treatment is that of the condition of malnutrition present.

HAY FEVER.—This is a condition in which some constitutional peculiarity of the individual causes an abnormal sensitiveness of the mucous membrane to some particular external irritant—usually the pollen of some plant. A nasal discharge, occurring periodically, and persisting for several weeks, may be the chief symptom of this irritation.

EPISTAXIS

During the period of early childhood epistaxis, or hemorrhage from the nose, is not uncommon. I have occasionally met with epistaxis in young infants, but in my experience it is rare in the early months of life. In older children recurrent epistaxis, especially if unilateral, points to the presence of an erosion or a varicose condition in the cartilaginous septum near the external opening of the nose.

Unless the individual happens to be affected by hemophilia, epistaxis is not especially dangerous, and usually its occurrence ceases as the child grows older.

TREATMENT.—The application of pressure on the side of the base of the nose and the use of ice are usually sufficient to stop the hemorrhage. If the epistaxis is due to the varicose condition just spoken of, it can be readily controlled temporarily by a plug of cotton pressed upon the bleeding part. For a permanent cure, cauterizing the bleeding part may be necessary.

III. DISEASES OF THE PHARYNX

ACUTE INFECTIONS

The term *angina* is often used to describe an acute inflammatory disease of the lymphoid ring of the pharynx, of infectious origin. The term is a poor one, as it means nothing more than pain, but is a useful abbreviation for these infections. Pharyngeal inflammation is seen in quite a number of recognized infectious diseases of known etiology, and is the principal lesion of two specific infections, scarlet fever and diphtheria. It is seen at times in other exanthemata, and in influenza, pneumonia, typhoid fever, etc. In addition to being a part of these infections, angina frequently represents a primary disease of varying bacteriological etiology.

GENERAL ETIOLOGY.—The causes of these infections of the lymphoid ring of the pharynx are of two kinds, predisposing and exciting.

PREDISPOSING CAUSES.—The various causes predisposing to these infections of the throat act by producing conditions favorable to bacterial development. There is no doubt that certain children have a constitutional predisposition to angina, and suffer from repeated attacks upon the least provocation. Such a constitutional predisposition may be inherited, or acquired through faulty hygiene. Hereditary constitutional predisposition, the so-called diathesis, appears to play a particularly important part in the acute infections of the pharynx, and a tendency to sore throat is closely connected with a tendency toward arthritis, carditis, and other so-called rheumatic manifestations. If a tendency toward such manifestations may be called a rheumatic diathesis, then we can undoubtedly say that angina is particularly common in this diathesis. Angina may, in this connection, be considered as especially important, as being the probable portal of entry for the virus of rheumatic fever, and possibly of epidemic meningitis and other specific infections. The constitutional predisposition toward " sore throat " often disappears after the age of puberty.

Certain local conditions in the throat have been associated with angina as predisposing causes. Among these are adenoids and chronic hypertrophy of the tonsils. While these conditions may predispose to " sore throat " in certain cases, many children with hypertrophied tonsils are free from " sore throat " while children with apparently normal tonsils may be subject to repeated attacks. Of the two, more weight must be attached to constitutional predisposition, and indeed, insofar as susceptibility to bacterial invasion is concerned, the so-called diatheses are constantly assuming more and more importance in the currently held views of the mechanism of infection.

Exposure to cold, and acute digestive disturbance, apparently act in certain cases as immediately predisposing causes. They act by producing a sudden lowering of the normal resistance against bacterial invasion.

EXCITING CAUSES.—The immediate cause of acute inflammation of the lymphatic ring of the pharynx is infection. No organism which has not been found in normal throats has been identified with this disease. The organisms most frequently associated with severe attacks are the *streptococcus*, the *pneumococcus*, the *staphylococcus*, the *micrococcus catarrhalis*, and rarely, the *bacillus of influenza*. In milder attacks the bacterial picture is often not clear. Mixed infections are probably common, and there is probably great variation in the virulence of the causative microörganisms.

In the majority of cases not occurring in a recognized epidemic of septic sore throat, the invasion of the tissues of the throat with pathogenic microörganisms probably occurs through autoinfection. Nevertheless, there is evidence that the infection can be transmitted from one affected individual to another. Family and institutional epidemics have been reported, and instances of apparent contagion are not uncommon.

VARIETIES AND CLASSIFICATION.—Owing to the varied and mixed bacteriology of angina, an etiological classification upon the basis of bacteriology is difficult. Nevertheless, there has been shown to be a certain amount of correspondence between particular bacterial infections, and the lesions produced, which has made it possible to separate off certain definite disease entities on an etiological basis. For instance, the cases of membranous tonsillitis not due to diphtheria have been shown to be almost invariably streptococcus infections. Again, there occur at times definite epidemics of infectious sore throat without constant lesions, but also due to some variety of streptococcus. This form is sufficiently definite in etiology to warrant its conception as a distinct disease, and it will be described under the name of epidemic septic angina. Another form of angina has not only a specific organism, but also a characteristic lesion. This form will be considered under the name Vincent's Angina.

The remaining cases of angina, constituting the majority, show neither a correspondence between bacteriology and lesions, nor any definite epidemic tendency. It is probable that many of them represent mixed infections. These can only be classified upon the basis of their clinical manifestations.

The infections of the lymphatic ring of the pharynx can therefore be classified as follows:

A. Variable or mixed infections:
    1. Acute Pharyngitis.
    2. Follicular Tonsillitis.
    3. Retronasal Angina.
B. Specific infections:
    1. Membranous Tonsillitis.
    2. Epidemic Septic Angina.
    3. Vincent's Angina.

ACUTE PHARYNGITIS

In this form the inflammation usually involves the entire mucous membrane of the fauces, including the pillars, tonsils, uvula, and the walls of the pharynx. Any part of the mucous membrane may, however, be involved alone, particularly the posterior wall of the pharynx. There is first acute redness and dryness of the mucous membrane, which may subside after twenty-four hours, but which is usually followed by swelling of the mucous membrane, and hypersecretion of mucus.

SYMPTOMS.—Sore throat is the principal symptom complained of. There is pain on swallowing, and a sensation of dryness in the pharynx. There may be a dry hacking cough. There is usually but little constitutional disturbance, but occasionally, in young children, the disease may have a rather violent onset with vomiting and a rise of temperature which may even reach 104°.

On examination of the throat, a reddening of the mucous membrane is seen, without any exudate other than a viscid mucus. The tonsils may be swollen as well as reddened. There may be slight swelling of the cervical lymphnodes.

The symptoms are usually of short duration, often subsiding in twenty-four hours, and rarely lasting more than two or three days. In some cases extension of the infection may produce an accompanying or following laryngitis.

DIAGNOSIS.—Scarlet fever, measles and diphtheria are the principal diseases to be excluded. *Scarlet fever* is attended by more severe constitutional disturbance than the average case of primary acute pharyngitis. In many cases, particularly those cases of acute pharyngitis having a severe onset, scarlet fever cannot be excluded until a sufficient time has passed for the appearance of the eruption. Consequently, if scarlet fever is prevalent, suspicious cases of sore throat should be isolated for forty-eight hours. *Measles* can usually be excluded more easily, by the absence of Koplik's spots on the mucous membrane of the mouth, and by the absence of coryza and lachrymation. *Diphtheria*, while usually showing a membranous lesion, may in rare cases, give only catarrhal manifestations in the throat; such cases can only be excluded by culture.

PROGNOSIS.—Recovery is usually rapid and complete.

TREATMENT.—At the beginning of the attack, the bowels should be freely opened. Magnesia or rhubarb and soda may be used for this purpose. One to three grains each of powdered rhubarb and bicarbonate of soda are given to children from one to six years of age. If there is vomiting, the magnesia may be preceded by one or two grains of calomel in divided doses.

The child should be kept in bed, on a liquid diet. In infants the quantity of food should be reduced. The child may dissolve pieces of ice in the mouth for relief of pain.

No internal medication need be given, and no local treatment of the throat should be used in infants, and in children too young to gargle. Older children will often get relief from the pain of swallowing by using hot gargles. Seiler's solution may be used, or a simple solution of a teaspoon of cooking soda to a glass of hot water. The important point is that the gargle should be as hot as can be borne.

FOLLICULAR TONSILLITIS

In this form the principal lesion produced by the infection is swelling and inflammation of the lymph follicles of the tonsils, with exudation into the tonsillar crypts. While the principal lesion seen involves the tonsils, there is also usually swelling and inflammation of the follicles of the entire pharyngeal ring. The exudation in the crypts consists of epithelial cells, pus cells, mucus, bacteria, and sometimes fibrin. The process is almost always bilateral.

SYMPTOMS.—The disease is characterized by an acute onset, with marked constitutional disturbance. The temperature rises rapidly, and is usually high, 103° to 105° F. In infants there is loss of appetite, frequently vomiting, and sometimes diarrhea. In older children there may in addition be chilly sensations, or even a distinct chill, at the onset, followed by a general malaise, with headache, and pain in the back and limbs. The general symptoms precede the local ones, and give the impression of some rather severe acute infection. Later, older children complain of sore throat, but the local discomfort is apt to be much slighter in children than in adults. In infants, there are usually no symptoms pointing toward the throat.

On examining the throat, the tonsils are seen to be enlarged, reddened, and in the early hours of the disease to show a little swelling of the orifices of the crypts, as though a secretion within them was about to burst the overlying mucous membrane and appear on the free surface. Later this actually occurs, and the tonsils are seen to be studded with yellowish or grayish-white spots. These do not appear on the soft palate or uvula, though they may appear on the pharyngeal tonsil, the base of the tongue, and the posterior pharyngeal walls. As the disease progresses these spots may coalesce and, adhering to the surface of the tonsil, form larger spots of exudation. The mucous membrane of the pillars of the palate, of the uvula, and of the soft palate are usually reddened, and there is usually decided reddening and swelling of the follicles of the pharynx. As there is a direct connection between the tonsils and the cervical glands, the latter are liable to be involved, though any great swelling of the cervical glands in connection with acute tonsillitis is uncommon.

The disease is self-limited and runs its course in two or three days, at the end of which time the general symptoms subside, the appetite returns, the temperature becomes normal, although the local symptoms may last somewhat longer.

DIAGNOSIS.—The lesion of tonsillitis is so characteristic, that the existence of multiple discrete spots of exudate on both tonsils is usually sufficient to warrant a diagnosis. At the onset scarlet fever cannot be excluded at times. Diphtheria can never be excluded without a bacteriological examination, and a culture should be taken from every case showing exudate on the tonsils. In the great majority of instances, however, when the attack is acute, with high temperature, when the cervical glands are not especially involved, when the white spots on the tonsils are clearly located in the orifices of the crypts, and when there is no appearance of a membrane on the soft palate or pharyngeal wall, we can make a provisional clinical diagnosis of follicular tonsillitis with considerable confidence.

PROGNOSIS.—The prognosis of follicular tonsillitis is favorable, the only important complication being peritonsillar abscess.

TREATMENT.—At the onset of the disease the child should be given castor oil, or, if there is vomiting, one or two grains of calomel in divided doses, followed by a saline. The child should be kept in bed, and isolated from all members of the household, except those having its immediate care. In infants, the milk should be diluted, and in older children, a milk and cereal diet should be used during the most acute stage. Ice cream is soothing to the throat, and may be given to older children.

TREATMENT OF INFANTS.—Infants rarely require symptomatic treatment, and most of the drugs generally recommended in tonsillitis such as quinin, aspirin, and sodium salicylate, are liable to upset the stomach. Local treatment also is unnecessary. I believe, however, that the internal administration of chlorate of potash is of value, especially in infants, given in frequent doses, as described under stomatitis. The amount to be given to children of different ages is shown on page 99.

TREATMENT OF OLDER CHILDREN.—Older children should be given aspirin or sodium salicylate, in doses of three to five grains every four hours for the first twenty-four hours, and three times a day after that. Hot gargles will add to their comfort. Seiler's solution, Dobell's solution, or a solution of sodium borate may be used. Painting or swabbing the tonsils should be avoided. Chlorate of potash should be given internally, to older children as well as to infants. If the throat is very sore, or if the cervical lymphnodes are painful, cold compresses should be applied to the throat.

RETRONASAL ANGINA

This form in etiology, and in the character of the lesion, represents exactly the same process as follicular tonsillitis. The difference is that the tonsils are not involved, and consequently no spots being seen on the tonsils, the disease is usually considered as being a severe rhinitis. In reality, the disease is a tonsillitis of the "third tonsil," or adenoid, and if posterior rhinoscopy is possible, the pharyngeal tonsil will be seen to be acutely inflamed.

SYMPTOMS.—The onset and general symptoms are much like follicular tonsillitis, with high temperature, and marked constitutional disturbance. Difficult breathing, with nasal voice are the suggestive symptoms in this form, rather than difficulty in swallowing. There is often earache, or deafness. There is usually a profuse mucopurulent discharge from the nose, and much mucus will be seen adherent to the posterior wall of the pharynx, which appears reddened and of uneven surface. The swollen adenoid may be felt by the finger, or seen by posterior rhinoscopy; this is, however, usually impossible in young children. The cervical lymph glands are more apt to be swollen than in follicular tonsillitis.

This disease is apt to run a more prolonged course than follicular tonsillitis. Nervous symptoms are more common, and I have seen this condition diagnosed as meningitis. The fever is sometimes remittent or intermittent, and the cervical lymphnodes may remain swollen for a considerable time, with prolonged elevation of temperature.

DIAGNOSIS.—In the early stages, it is difficult to distinguish acute infection of the adenoid from the common acute rhinopharyngitis. Posterior rhinoscopy would reveal the nature of the disease, but is difficult in young children. Often retro-nasal angina can only be suspected from the severity of the constitutional disturbance, and the persistence of the symptoms.

PROGNOSIS.—The prognosis is favorable, but the course of the disease is apt to be more prolonged than in either acute rhinitis or follicular tonsillitis.

TREATMENT.—The treatment is that of acute rhinitis. In older children, hot gargles may be used in addition.

MEMBRANOUS TONSILLITIS

A membranous tonsillitis occurs most frequently as a secondary manifestation in an acute infectious disease, especially in scarlet fever and measles. Cases are found, however, in which membranous tonsillitis occurs as a primary inflammation. The bacteriology of membranous tonsillitis is sufficiently constant to warrant its being classed as a separate disease entity.

ETIOLOGY.—The inflammation is almost invariably due to a streptococcus. In some cases a staphylococcus has been found associated with the streptococcus, and in very rare cases, the staphylococcus has been found alone.

Infection probably occurs in most cases through autoinfection. Streptococci are frequently found in the throats of healthy children. Any condition lowering the resistance of the tissues can probably act as the cause of the occurrence of infection. In the acute infectious diseases, not only may the general resistance be lowered, but the local conditions in the mucous membrane are especially favorable to secondary infection. In the primary cases, a condition lowering the resistance is often not found. It is possible that these cases are infections from without, with streptococci of especial virulence, but there is little evidence of transmission by contagion. While transmission is possible, I believe the usual method is through autoinfection.

PATHOLOGICAL ANATOMY.—The essential lesion is the formation of a pseudo-membrane, much resembling that seen in diphtheria. In structure the membrane is indistinguishable from that found in diphtheria. It may be superficial or deep. It is usually confined to the tonsils, or is situated chiefly on the tonsils, but may extend to a variable extent to all the mucous membrane of the pharynx, the soft palate, uvula, and may in rare cases extend to the mouth, larynx, bronchi, nose, and ear.

The streptococci are found in and under the membrane, and in the lymph spaces. There is an accompanying acute inflammation of the lymphoid tissue of the pharynx.

SYMPTOMS. THE PRIMARY CASES.—The onset and general symptoms of primary membranous tonsillitis are precisely like those of follicular tonsillitis. Pain on swallowing is apt to be more marked. There is reddening and swelling of the tonsils, and often of the tissues of the pharynx. Patches of membrane appear on the tonsils, usually of irregular outline, and not sharply defined. The membrane appears loosely attached, and can sometimes be wiped off. In color the membrane is yellowish or greyish, and occasionally greenish. Usually the membrane is limited to the tonsils, and does not spread, but occasionally small patches are seen on other parts of the mucous membrane. The course of the disease is like follicular tonsillitis.

The Secondary Cases.—In general, these cases are more severe, although mild cases are seen resembling the primary form. Usually they are characterized by high fever and great prostration. The nervous system is often profoundly affected, there being apathy, delirium or stupor. The pulse is usually rapid and feeble. The membrane may be much more extensive than in the primary cases. Complications frequently seen are bronchopneumonia and nephritis.

DIAGNOSIS.—The only question in cases showing membrane on the tonsils is whether it is a streptococcus infection or diphtheria. The clinical features distinguishing the two conditions have been described under diphtheria, but a diagnosis based on clinical grounds is most uncertain. Cultures should be taken in every case showing membrane in the throat.

PROGNOSIS.—The primary and mild cases run a short course resembling that of follicular tonsillitis, and the disease is not serious. The secondary cases are much more serious. The course is longer, and death may occur from exhaustion, or from one of the complications. When a complication occurs, the prognosis is very grave. If no complication occurs, in spite of the severe toxemia, and no matter how severe the symptoms, there is a good chance of recovery.

TREATMENT.—Every case should be isolated as soon as the membrane is seen. The question which first confronts the physician is whether he shall give diphtheria antitoxin at once, or wait for the result of the examination of the cultures. I believe that in cases secondary to another already recognized infection, and in older children, one can wait for the result of bacteriological examination. In primary cases in children under three years, antitoxin should be administered at once without waiting.

The treatment of the primary and mild secondary cases is that of follicular tonsillitis. The treatment of the severe cases is with the exception that antitoxin is not given, the same as that of diphtheria.

EPIDEMIC SEPTIC ANGINA

Epidemics of severe sore throat have been reported from time to time in various parts of the world. The constancy of the bacterial findings and of the mode of infection in these epidemics, and certain distinctive clinical features, warrant the conception of a distinct disease.

ETIOLOGY.—The microörganism found in the various epidemics is a streptococcus. The type of streptococcus, wherever studied, has shown the same peculiarities distinguishing it from other streptococci.

The usual mode of infection is through *milk*. All of the epidemics studied have been milk epidemics, traceable to a definite milk supply. Such milk epidemics of septic sore throat have been reported with frequency in England, and occasionally in other parts of Europe, but have only recently been observed in this country. While milk is the usual means of transmission of the infection in epidemic sore throat, and while milk epidemics have been the only ones studied bacteriologically, it is probable that epidemics of infection with the same organism may occur, without transmission by means of milk. It is notable that during and preceding the Boston milk epidemic of 1911, there was an outbreak of severe tonsillitis in the regions from which the milk came, but in which the milk was not used. The clinical features were the same as those observed in the Boston cases. In this outbreak, which could not be traced to milk, the means of transmission may have been by contagion. Milk is, however, probably the usual disseminator of the disease.

The source of the contamination of the milk supply is still an unsettled problem.

Adults are more frequently affected with epidemic sore throat than are children. This was a notable feature of the Boston epidemic of 1911. I have seen a smaller epidemic in which children were affected with the same frequency as adults.

SYMPTOMS.—The distinctive clinical features of epidemic septic angina are, the severity of the sore throat and of the constitutional disturbance, the lack of constancy of appearance in the throat, the constancy of involvement of the cervical lymphnodes, and the tendency to metastatic infection in remote parts of the body.

The period of incubation in the Boston epidemic was short, not more than thirty-six to seventy-two hours. The onset is sudden, with sore throat, and often with chills, headache and prostration. The fever is apt to be moderate in proportion to other signs of constitutional disturbance. Nausea and vomiting are common, especially in children, in whom the gastro-intestinal symptoms are often more noticeable than the throat symptoms.

The local appearance of the throat varies. In all instances the primary infection occurs in the lymphoid tissue of the fauces and pharynx, but the local pathological alterations show a wide range, from acute congestion and swelling of the lymphoid tissue, to infiltration of the adjacent mucous membrane and circumtonsillar suppuration. The most common appearance is that of follicular tonsillitis; the mildest cases resemble acute pharyngitis in local appearances, while in severe cases the lesions are those of membranous tonsillitis, often with marked edematous swelling of the uvula, soft palate, and base of the tongue.

The disease is apt to be milder in children than in adults. Mild cases show improvement in two or three days, and are convalescent in a week.

Severe cases, while more common in adults, may occur in children. The temperature in these cases may reach 104°, with very marked constitutional disturbance, rapid pulse, severe pain in the head, back and extremities, insomnia or mild delirium, and very marked prostration. In these cases relapses are frequent, prostration is persistent, and convalescence slow.

In all but the mildest cases there is an involvement of the cervical lymphnodes, which become enlarged, and often intensely painful.

A marked feature of the disease is the tendency toward complications of various kinds, mainly metastatic septic processes. More than one-quarter of the cases in the Boston epidemic had secondary processes. Among the complications frequently seen are the following: Peritonsillar abscess, suppuration of the cervical lymphnodes; arthritis, peritonitis, pleurisy, and pericarditis; pneumonia, laryngitis; endocarditis, phlebitis, and septicemia; acute nephritis.

DIAGNOSIS.—The diagnosis cannot be made in any single case, but is based on the epidemic character of the disease, and upon the occurrence of other cases having the same milk supply. In every case of severe or atypical tonsillitis, efforts should be made to determine if similar cases have been observed in the vicinity.

PROGNOSIS.—The disease is serious, from the tendency toward severe septic complications. The mortality in the Boston epidemics was 48 cases out of a total of 1,043. The prognosis is better in children than in adults.

TREATMENT.—Mild cases of epidemic sore throat should be treated like cases of follicular tonsillitis. Severe cases require stimulation, and the general and local treatment should be like that of a severe case of diphtheria, except that antitoxin is not given. I believe that in severe cases, when the particular streptococcus can be isolated in cultures, an autogenous vaccine should be made and tried.

In prophylaxis, pasteurization of the milk given to the child will prevent infections of this character. It is noteworthy that the milk supply found to be responsible for the Boston epidemic of 1911 was considered to be one of the very best, the milk being produced under the cleanest possible conditions. The occurrence of this epidemic is a strong argument in favor of the routine use of pasteurized milk in feeding infants and children.

PROBLEMS AND RESEARCH.—The principal questions arising in connection with epidemic sore throat are the following: 1, Are the streptococci causing different epidemics the same, representing a specific organism, or are different strains represented? 2. How does the milk become contaminated with the streptococci? 3. Are the streptococci of human or of bovine origin? Various studies of recent epidemics have established the fact that the streptococci in various epidemics all have the same cultural characteristics which distinguish them from common throat streptococci. An elaborate study of the streptococci from a number of different outbreaks was made by Theobald Smith and J. H. Brown. They concluded that the streptococci are not necessarily the same in various outbreaks, but that they differ in certain minor distinguishing characteristics, and that a detailed study of individual strains is necessary in order to trace an epidemic to its source.

In connection with the second question, the epidemiology of most of the reported epidemics has been of such a character, as to suggest that the contamination of the milk cannot be explained by transmission of the organism directly from an infected human individual to the milk. It rather suggests disease in the udder of some cow. Cow mastitis or garget is a comparatively common condition. Is this the source of the epidemic?

In connection with the third question, various experiments have been made with streptococci from spontaneous cow mastitis, and with the production of experimental cow mastitis. The results of these experiments are summarized by Smith and Brown, and seem to show that the streptococci of the cow mastitis or garget are different from the streptococci of human tonsillitis, that the virulent streptococci of man do not cause any appreciable inflammation of the cow's udder, and vice versa, that the mastitis streptococci do not cause throat affections in man. Nevertheless the udder of a cow inoculated with virulent human streptococci may permit certain strains to multiply in the milk ducts, and thus be shed into the milk for a considerable time. Smith and Brown believe this to be the probable source of the contamination, accidental inoculation of the udder with virulent human streptococci.

VINCENT'S ANGINA

(Ulcero-membranous Tonsillitis.)

This is a specific infectious disease, caused by the same organism as is seen in ulcerative stomatitis, and characterized by an ulcero-membranous lesion of the tonsil.

ETIOLOGY.—This disease is caused by an organism known as the bacillus fusiformis, with which is almost always associated a spirillum (or spirocheta). In typical lesions the bacterial picture is very constant, both the bacilli and spirilli being very numerous. The bacillus varies greatly in length, measuring from four to sixteen micra in length, and about one-half micron in thickness, with tapering ends. It stains readily with all ordinary stains, the staining being somewhat irregular. The spirillum is longer than the bacillus, very delicate, and stains less strongly.

The evidence in favor of the specific nature of the infection is based on the constancy of the bacterial picture, and on the enormous number of these organisms found in typical lesions. While other varieties of microörganisms are found in the superficial parts of the lesion, the deeper layers show appearances suggesting a pure culture. With this bacterial picture goes a marked constancy of anatomical lesion. The organisms regularly disappear as the healing process begins.

The usual mode of infection is probably by autoinfection. The organisms have been shown to be frequently present in normal mouths, and in other lesions of the mouth, such as ulcerative stomatitis, noma, pyorrhea alveolaris, and are found occasionally in gangrenous processes in other parts of the body. Most authorities believe that some injury to the mucous membrane is an antecedent factor permitting the occurrence of infection. There have been apparent instances of transmissibility by contagion, but this feature has not been marked. There have been numerous instances of transplantation from one part of the mouth to another.

The disease affects both children and adults, but children are much more frequently affected.

PATHOLOGICAL ANATOMY.—The lesion is an ulcer, with irregular sharp cut edges, filled with granular necrotic material having a putrid odor. These ulcers occur by a process of necrobiosis. There is remarkably little inflammatory reaction surrounding the ulcer, either none, or a very narrow zone. Occasionally there is more surrounding redness and swelling, but this is probably due to some complicating organism.

The lesion may be situated on any part of the mouth, but is usually found on the tonsil, and is unilateral. It may extend to the adjacent posterior pillar, or to the edge of the uvula.

SYMPTOMS.—The onset is usually gradual, and sometimes the disease is first detected in the child from the foul odor of the breath. There may be moderate fever, 99° to 102°, and slight malaise, but constitutional disturbance is usually slight. Sore throat is usually complained of, but is never severe, and seems very slight in comparison with the lesion seen on examining the throat. The appetite is impaired, and older children complain of a bad taste in the mouth.

On examination the breath is markedly foul. On one tonsil usually, is seen an ulcer, with sharp-cut edges, and a ragged looking base, which in color is yellowish, greyish, or greenish. At times the loss of tonsillar substance may be considerable, as much as half the tonsil being replaced by a greyish crater. If the granular necrotic material be removed from the base of the ulcer, a bleeding irregular surface is seen.

There is usually little or no redness and swelling surrounding the ulcer. The cervical lymphnodes are occasionally enlarged, but are usually not tender.

Occasionally the ulcers tend to enlarge rapidly, but usually enlargement is slow, or absent. Without treatment, the ulcers may persist for a period varying from a few days to several weeks, according to the general condition of the nutrition and the hygienic surroundings of the child. With treatment, the ulcers heal in a few days.

DIAGNOSIS.—A fairly accurate diagnosis can be made from the clinical appearances alone. The disease is most frequently mistaken for diphtheria, but the character of the lesions in the two diseases is entirely different. In diphtheria and membranous tonsillitis the membrane appears to be plastered on the surface of the tonsil, and there is no evident loss of tonsillar substance, whereas in Vincent's angina, there is always loss of substance, the membrane occupying a concave crater. Also, in diphtheria and membranous tonsillitis there is redness and swelling of the surrounding mucous membrane, which are notably absent in Vincent's angina. The lesion of the disease is of such a character that it cannot be mistaken for follicular tonsillitis.

The diagnosis can be definitely settled by making a microscopic examination of the necrotic material at the base of the ulcer. This should be wiped off, and spread on a coverglass, and stained with carbol-fuchsin or methyl-violet, which will show the spirilli as well as the fusiform bacilli. It should be remembered that mixed infections are always possible, and diphtheria cannot be positively excluded, even if cover-glass preparations show the bacteriology characteristic of Vincent's angina. Cultures for diphtheria should always be taken, especially if there be any surrounding inflammatory reaction.

PROGNOSIS.—The prognosis is favorable. With treatment, healing is rapid.

TREATMENT.—Potassium chlorate acts almost as a specific in Vincent's angina. It should be given internally in frequent doses, in the way recommended for ulcerative stomatitis. No other treatment is necessary.

PROBLEMS AND RESEARCH.—Vincent's angina, and the various other ulcerative and gangrenous conditions in which the same bacterial picture is found, present a most fascinating bacteriological problem. The principal question is the relation of the two organisms, the fusiform bacillus, and the spirillum, to one another. There is a marked divergence of opinion on this point. Some observers believe the two organisms to be different, and that their association is an example of symbiosis. It has been shown that in cultures the bacillus fusiformis grows better when associated with contaminating organisms. It is a strict anaerobe, and may be grown best on fresh blood serum from animals, to which has been added sugar bouillon.

Other observers believe that the two organisms represent different stages of the same organism. It has even been suggested that the organism is not a bacterium, but an animal parasite closely related to the trypanosomes. Tunnicliff has reported cultural studies of great interest, in which apparently pure cultures of the bacillus developed into spirillum forms. Nevertheless, the difference in morphology is so great, that one cannot accept so remarkable a transfiguration without much more evidence.

Another point of interest is the relation of infection with these organisms to other infections—whether Vincent's angina represents a primary infection, or whether it is preceded by some other infection, which is overlooked at the start, and later overshadowed.

RETROPHARYNGEAL ABSCESS

There are two varieties of abscess seen in the tissues of the posterior pharyngeal wall. One is a comparatively rare complication of tuberculosis of the cervical vertebrae, and is usually seen in older children. The other is the so-called idiopathic form of infancy. Only the latter will be considered here.

ETIOLOGY.—Streptococci are usually found in these abscesses, but mixed infections are common, both staphylococci and influenza bacilli being sometimes found with the streptococci. The process is never primary, but represents an extension of infection from some part of the throat or naso-pharynx. The most common primary diseases are rhino-pharyngitis, retronasal angina, or an attack of epidemic influenza. The disease may occur as a sequel of measles, scarlet fever, or diphtheria.

It is almost exclusively a disease of infancy, and is seen most frequently in the first year of life. This is due to the abundance and functional activity of the lymphnodes in the pharyngeal wall at this age, and their connection with the lymphnodes of the nasopharynx.

PATHOLOGICAL ANATOMY.—The inflammatory process is not primarily a cellulitis, but a lymphadenitis. There is a chain of lymphnodes on either side of the median line, between the pharyngeal and prevertebral muscles. One or more of these nodes is the starting-point of the process. At first there is inflammatory swelling of the node, then periglandular infiltration, then suppuration, beginning in the node. The pathology is exactly the same as suppuration of the external cervical lymphnodes. The inflammation may subside without suppuration taking place.

SYMPTOMS.—The first symptom is generally difficulty in swallowing, which may go on to entire inability to swallow. The infant is next noticed to breathe in a peculiar way. It holds its head back and its mouth open. The breathing may be described as snorting, and at times is almost stertorous, differing markedly from the whistling sound which is heard in obstruction of the larynx. The voice is nasal and there is often a peculiar short cry. On examining the throat the soft palate is seen to be pushed forward and to be somewhat anemic. The posterior wall of the pharynx is bulging, usually unilaterally, is reddened, swollen, tense, and as the disease progresses is found to be fluctuating. In some cases the swelling cannot be seen through the mouth and can only be detected by palpation.

There is usually some elevation of temperature. The onset is apt to be insidious, the symptoms often developing gradually after an attack of rhinitis, or infectious angina. There may be prostration and loss of weight, and quite high temperature may be reached, 102°-104^{c}. In other cases constitutional symptoms are slight.

DIAGNOSIS.—Whenever in any infant is seen difficulty in swallowing, mouth breathing, noisy snorting dyspnea, or the head drawn back, retro-pharyngeal abscess should be suspected. The diagnosis can only be confirmed by a thorough digital exploration of the pharynx. These cases are often not seen by the physician until the dyspnea has attracted attention. In this stage I have often seen the condition mistaken for laryngeal obstruction. The snorting character of the respiration, with the characteristic position of the head are usually present in retropharyngeal abscess, but not always, and a digital examination of the pharynx should be made in these cases. In less marked cases, the symptoms may suggest that only a rhinopharyngitis is present, but even at this stage, digital examination will often reveal either a hard or a fluctuating swelling of the posterior pharyngeal wall.

PROGNOSIS.—In some cases only a lymphadenitis may be present, causing a swelling, but no fluctuation, and in such cases the swelling may subside, but this is rare, and usually the process goes on to suppuration. After an abscess has developed, if left to itself, it wlil eventually open into the pharynx, the pus being usually swallowed, but sometimes expectorated. In case of spontaneous rupture, the cavity may close rapidly by granulation, or it may refill. If the abscess ruptures suddenly, especially during sleep, the pus may be drawn into the air passages, and cause death from suffocation. Occasionally fatal cases have been seen from pressure of the abscess upon the larynx, or from edema of the glottis. The course of the disease is shortened, and the dangers lessened by early diagnosis and proper treatment. The most frequent cause of death is bronchopneumonia, occurring at any time subsequent to the opening of the abscess. The general mortality is about ten per cent.

TREATMENT.—Before suppuration occurs, the general treatment should be that of retronasal angina. Hot applications to the external throat may aid the " pointing " of the abscess. As soon as fluctuation is found, the abscess should be opened at once. The method which I have found satisfactory in the cases which have come under my observation is to have an assistant hold the infant sitting upright in the lap, with a blanket tightly pinned around it so as to prevent it from moving its arms. Another assistant should hold the head. The mouth should then be opened, a guarded bistoury should be introduced into the pharynx and the abscess punctured. As soon as the opening has been made, the bistoury should be removed quickly, and the infant's head should be immediately thrown forward and downward, so that the pus will be discharged from its mouth and not inspired into the larynx or swallowed. It is usually necessary after the operation to introduce the finger into the pharynx and to press the walls of the abscess, so as to empty any pus which may continue to collect there and also to keep the opening free. With this treatment, unless some complication should arise, the disease is usually cured in about a week.

Some operators prefer having the child placed in what is known as Rose's position, on its back with the head hanging over the end of the table.

PERITONSILLAR ABSCESS

(Phlegmonous Tonsillitis.) (Quinsy.)

As compared with adults, quinsy is a rare disease in early life.

ETIOLOGY.—The disease is never primary, but is secondary to some infection of the throat, usually to a tonsillitis. The organisms causing the abscess are the same as those causing follicular tonsillitis. Streptococci are usually found, with or without a mixed infection.

Certain children appear to have a constitutional predisposition to quinsy, undergoing a number of attacks.

PATHOLOGICAL ANATOMY.—The process begins as a severe parenchymatous inflammation of the tonsil, which may terminate in resolution, but which usually results in the formation of an abscess in the cellular tissue surrounding the tonsil.

SYMPTOMS.—The disease usually begins with the symptoms of follicular tonsillitis. After a few days, the exudate disappears from the tonsils, but the throat symptoms, instead of subsiding become aggravated. There is very severe pain in the throat, which is so increased by deglutition that swallowing may become impossible. The neck and jaws usually become stiff and painful on motion. There is usually a rather high, irregular fever.

On examining the throat in the early stage, the appearances do not seem to account for the severity of the pain. Later one tonsil becomes swollen, and then pushed toward the median line by the swelling in the tissues behind the tonsil, which also pushes the soft palate forward. The mucous membrane of the swollen area appears intensely reddened. In this later stage, in the course of a few days, fluctuation can be detected at some part of the swollen area.

DIAGNOSIS.—The intense pain, with unilateral swelling, are the points on which the diagnosis rests.

PROGNOSIS.—If left to itself the abscess will eventually rupture spontaneously, with marked relief of the symptoms. The prognosis is favorable.

TREATMENT.—The abscess should not be left to rupture spontaneously, but should be opened as soon as, but not before, a distinct point of fluctuation is detected. Before fluctuation is detected, frequent hot gargles should be used, and very hot or cold external applications, according to the sensations of the patient. Dissolving cracked ice in the mouth often gives relief.

ADENOIDS

(Hypertrophy of the Pharyngeal Tonsil.)

The term " adenoid " is used as an abbreviation when referring to a pathological condition. Strictly speaking it refers to the collection of lymphoid tissue in the vault of the pharynx, also called the pharyngeal or " third " tonsil. This tissue is normally present, and in this strict sense, all children have adenoids. As commonly used, the term refers to adenoid vegetations of the vault of the pharynx, caused by hypertrophy of the lymphoid tissue situated there. In young children, a certain amount of hypertrophy of the pharyngeal tonsil is so common as to be practically a normal condition. The term adenoid therefore has the further significance that it refers to hypertrophy sufficient to cause symptoms.

ETIOLOGY.—The etiological factors which have been associated with the presence of adenoids are the following: 1. Hereditary constitutional predisposition; 2. Mechanical irritants; 3. Infection.

Hereditary predisposition, the so-called lymphatic diathesis, is believed to play an important part, because sometimes whole families of children are seen affected with adenoids, whose parents suffered from the same condition in childhood. I do not believe, however, that the influence of heredity is very firmly established on this evidence, though it is possible. Adenoids are very common, more so than can be explained by any hereditary factor. There is a tendency to lymphoid overgrowth characteristic of early life, and seen in all children. This factor I believe to be more important than heredity. There is also great individual variation in the conformation of the naso-pharynx, which would lead to a varying degree of disturbance from adenoid hypertrophy in different children.

The mechanical irritants associated with adenoids are dust, cold air, and damp air. The association is based on the fact that adenoids are more common in damp changeable climates, that the pharyngeal tonsil is particularly exposed to the action of these irritants, and that they might produce the hypertrophy. I am very doubtful if they are more than causes predisposing toward infection.

Infection is probably the chief etiological factor connected with adenoids. The adenoid tissue takes part in such acute infections as rhinitis, pharyngitis, and retronasal angina, and becomes more hypertrophied with each attack. The adenoid tissue is a favorable place for the retention of the microörganisms which produce such attacks, holding them between attacks, so that a vicious circle is produced. I believe repeated infections are the principal cause of most cases of adenoid vegetations, although those cases in which adenoids are seen in very early infancy may be explained on the basis of tendency to lymphoid overgrowth plus a narrow pharyngeal vault.

Adenoid hypertrophy is almost exclusively a disease of childhood. It may be present at birth, or may occasionally develop during the first year. Usually, however, the symptoms do not develop until after the end of the first year, most cases developing between the eighteenth month and the fifth year, although they may not come under observation until a still later period of childhood.

PATHOLOGICAL ANATOMY.—The process is one of hyperplasia of the lymphoid tissue. The pharyngeal tonsil forms a tumor mass, which may be large enough to fill the rhino-pharynx completely. The tumor is attached to the vault of the pharynx by a broad base.

SYMPTOMS.—The symptoms of adenoids are very numerous, and vary much both in character and in severity. In general the symptoms are more marked in winter than in summer. They may be divided into three classes as follows: 1. Symptoms due to obstruction; 2. Symptoms due to inflammation of the mucous membranes; 3. Symptoms due to reflex irritation.

OBSTRUCTIVE SYMPTOMS.—The most constant obstructive symptom is mouth breathing. At first this may be seen only during sleep. The children sleep with their mouths open, and snoring is common. The obstruction to the breathing interferes with sleep, especially when the child is lying on its back. Sleep becomes restless, the child tossing about, and assuming a variety of strange positions. Later, the mouth breathing is seen also by day. The children hold their mouths open, and develop a characteristic facial expression. The adenoid face is not wholly due to the open mouth, but the masseter muscles become relaxed as a result of the constantly open mouth, so that the lower jaw is habitually dropped. This gives the face a stupid vacant look. In marked cases, the obstruction may give rise to actual developmental bony deformity. The lower jaw protrudes, the hard palate shows a high arch, and there may be some displacement of the teeth, while the bridge of the nose is flattened. This gives the impression of a long face, narrow toward the chin, with a prominent upper lip, and obliteration of the naso-labial folds. The chests of adenoid patients are usually poorly developed, but the bony deformity of the thorax often attributed to adenoids, I have only seen in rachitic children, and I believe the adenoid obstruction to be only one of several etiologic factors.

The voice of children with adenoids is often affected by the obstruction. It shows impaired resonance, and is sometimes nasal in character. Inability to blow the nose is usually seen in obstruction from adenoids.

INFLAMMATORY SYMPTOMS.—Adenoids greatly increase the tendency toward infections of the upper air passages. Children with adenoids are particularly liable to repeated head colds. Beside these frequent acute attacks, they have a chronic muco-purulent nasal discharge which is present at all times, though worse in winter. The attacks of acute infection of the upper air passages, while usually taking the form of an acute rhino-pharyngitis, may by extension take the form of pharyngitis, tonsillitis, laryngitis, or bronchitis. Catarrhal spasm of the larynx, or common croup, is frequently seen in young children with adenoids. A persistent cough, often spasmodic in character, is frequently present.

The hearing is more or less impaired in a large proportion of children with adenoids, and this deafness, which may not be sufficiently marked to be recognized by the parents, is often mistaken for defective mentality, this idea being strengthened by the adenoid

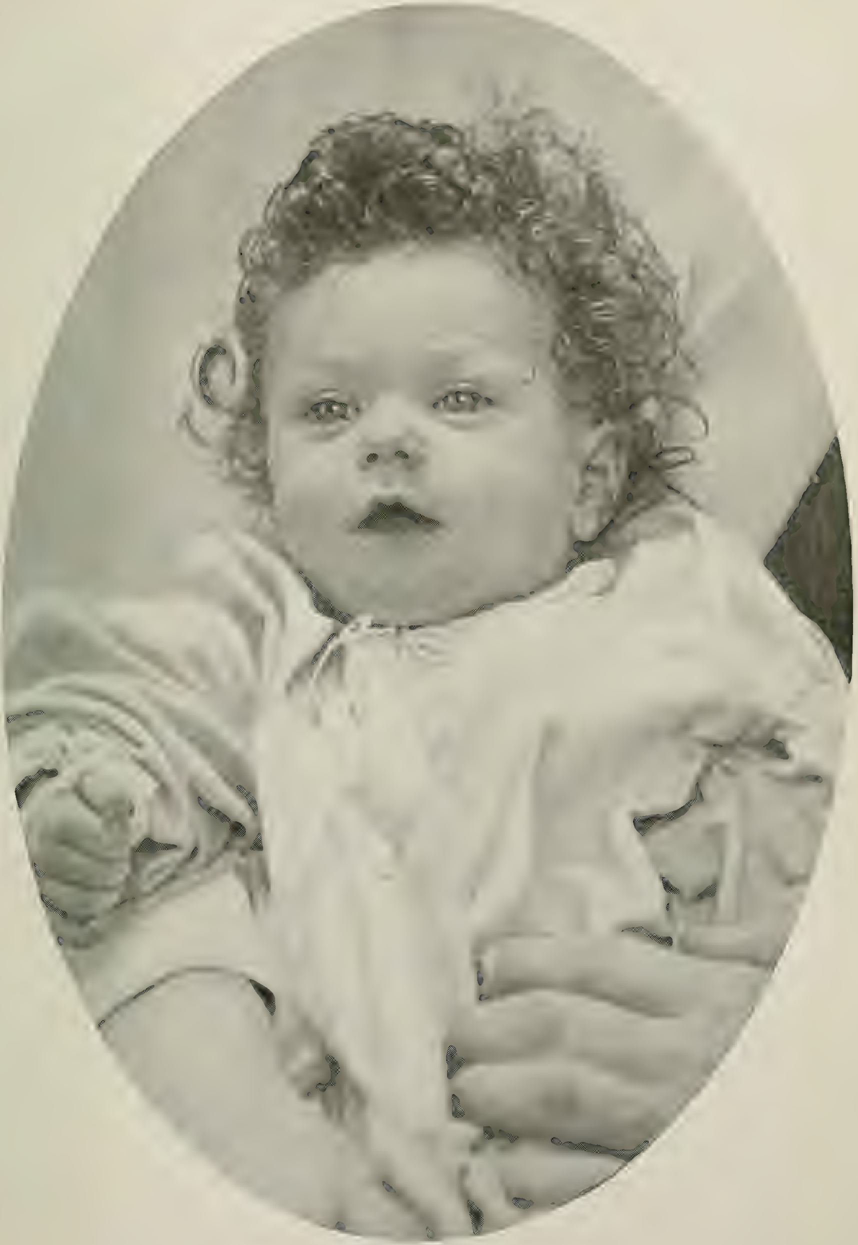


FIG. 232—Adenoid Facies in an Infant

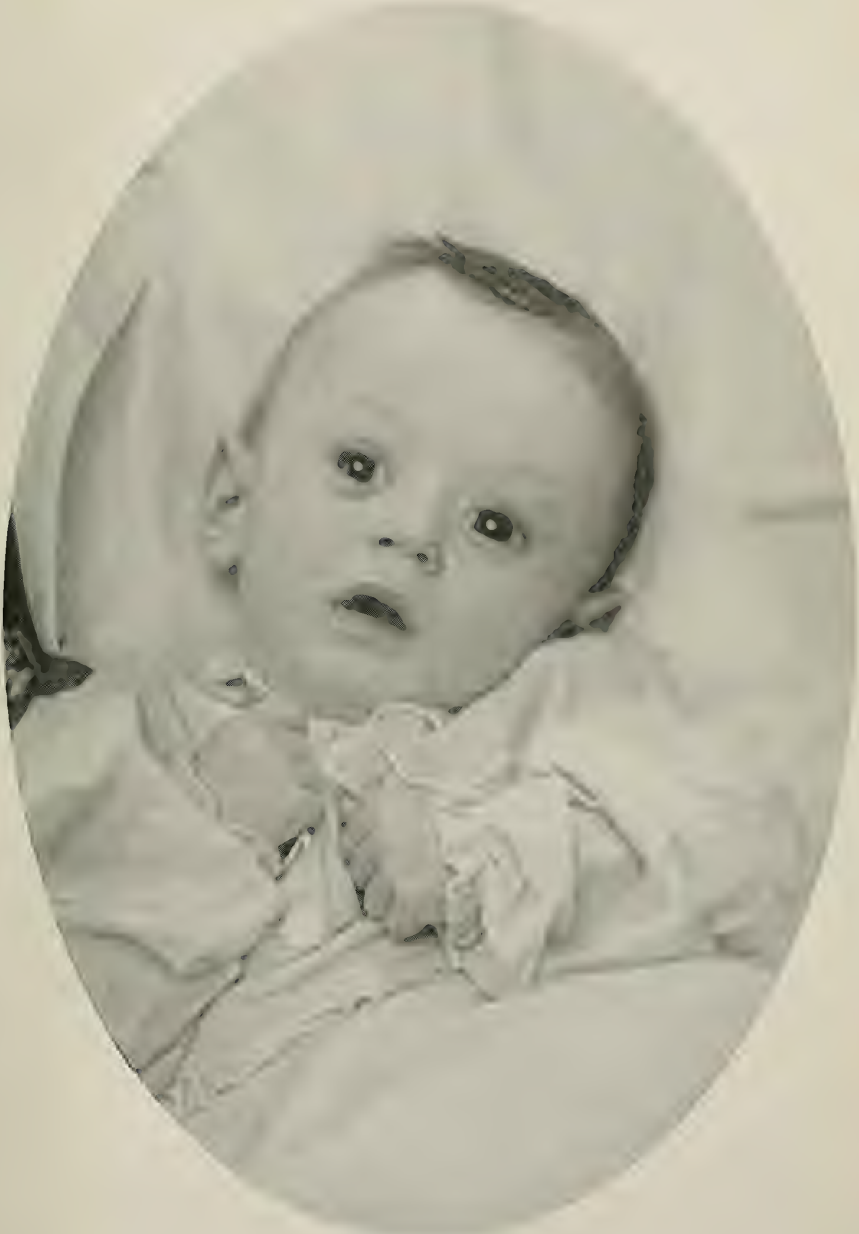
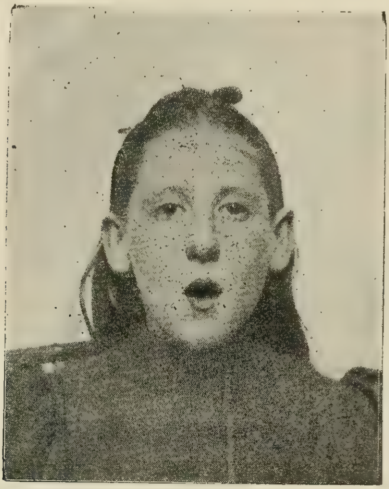


Fig. 233—Adenoid Facies in an Infant

facies. The deafness is due either to extension of the catarrhal inflammation into the Eustachian tubes, with swelling of the mucous membrane, closure of the tubes, deficient ventilation of the middle ear, and retraction of the tympanic membrane, or to extension into the middle ear, causing a chronic otitis media. Attacks of acute suppurative otitis media may also occur from time to time, and chronic discharging ears are not uncommon.

Chronic enlargement of the cervical lymphnodes, without tenderness, is a very common condition in children with adenoids.

FIG. 234



Hypertrophy of pharyngeal tonsil (adenoid growths). Female, 10 years old

REFLEX SYMPTOMS.—Children with adenoids are very liable to show increased nervous irritability. The connection with adenoids is shown by the fact that in children brought to the physician for "nervousness," the symptoms are often relieved after the adenoids are removed. Headaches are often seen in children with adenoids. Various distinct manifestations have been attributed to the reflex irritation from adenoids, the most important of which are enuresis, night terrors, disturbance of speech, and asthma. The evidence of the influence of adenoids is the relief sometimes seen after removal. I believe that reflex irritation is an important etiological factor in both enuresis, and night terrors. The latter condition is actually, in my opinion, a reflex phenomenon, and not due to beginning temporary suffocation from obstructed breathing, as it is still more common in a certain form of indigestion. I believe adenoids are a possible, though less common factor in stammering. I am very doubtful of any connection with asthma.

Children with adenoids are likely to be anemic and poorly nourished. The way in which adenoids cause impairment of the general health is not clear. It may be through lack of oxygen from obstructed breathing, or through disturbed sleep, but in my opinion the most probable cause is the confinement indoors necessitated by repeated colds. Many children with adenoids are languid, listless, and suffer from nervous depression.

DIAGNOSIS.—The history is usually sufficiently characteristic to enable the physician to suspect the presence of adenoids. The mouth breathing, disturbed sleep, chronic nasal discharge, frequent colds, and slight deafness form a well marked clinical picture. In those cases in which the child is brought to the physician for nervousness, anemia, malnutrition, enuresis, or night terrors, the possibility of adenoids must not be overlooked. The diagnosis can only be made sure when the vegetations are actually seen or felt.

The importance of learning to detect by means of the finger the presence of an enlarged pharyngeal tonsil is great. If an examination with the posterior rhinoscopic mirror can be made the exact extent of the hypertrophy can be seen, and the child spared the discomfort of a digital examination, but the examination with the mirror in the throat is usually so difficult in young children that the direct detection by means of the finger is often the best method. The child should have a blanket pinned around it tightly, so as to keep it from moving its arms. It should be held firmly sitting in the nurse's lap. The physician holds the child's head with one arm, pressing the cheek between the back teeth with the forefinger, then passes the forefinger of the other hand gently, firmly, and quickly over the base of the tongue and behind the soft palate until it reaches the posterior wall of the pharynx. By quickly turning the finger upward, it is easy to feel whether the cavity of the naso-pharynx is clear or whether it is more or less filled by a soft, spongy mass, the hypertrophied pharyngeal tonsil. There is usually a little blood on the finger when it is withdrawn, as the growth is friable and bleeds easily. In passing the finger over the base of the tongue it is necessary to be careful to get the finger behind the soft palate, and not to push it upward and backward, for in this case the soft tissues of the palate may feel like an adenoid growth. The child can be prevented from biting the finger by simply keeping the cheek pressed between the teeth as has just been described.

TREATMENT.—The only effective treatment for adenoids is removal by surgical operation.

The question which the physician has to decide is when operation is indicated. The mere presence of adenoid vegetations in the vault of the pharynx is not necessarily an indication for operation. Large adenoid growths may be present in certain cases, without giving

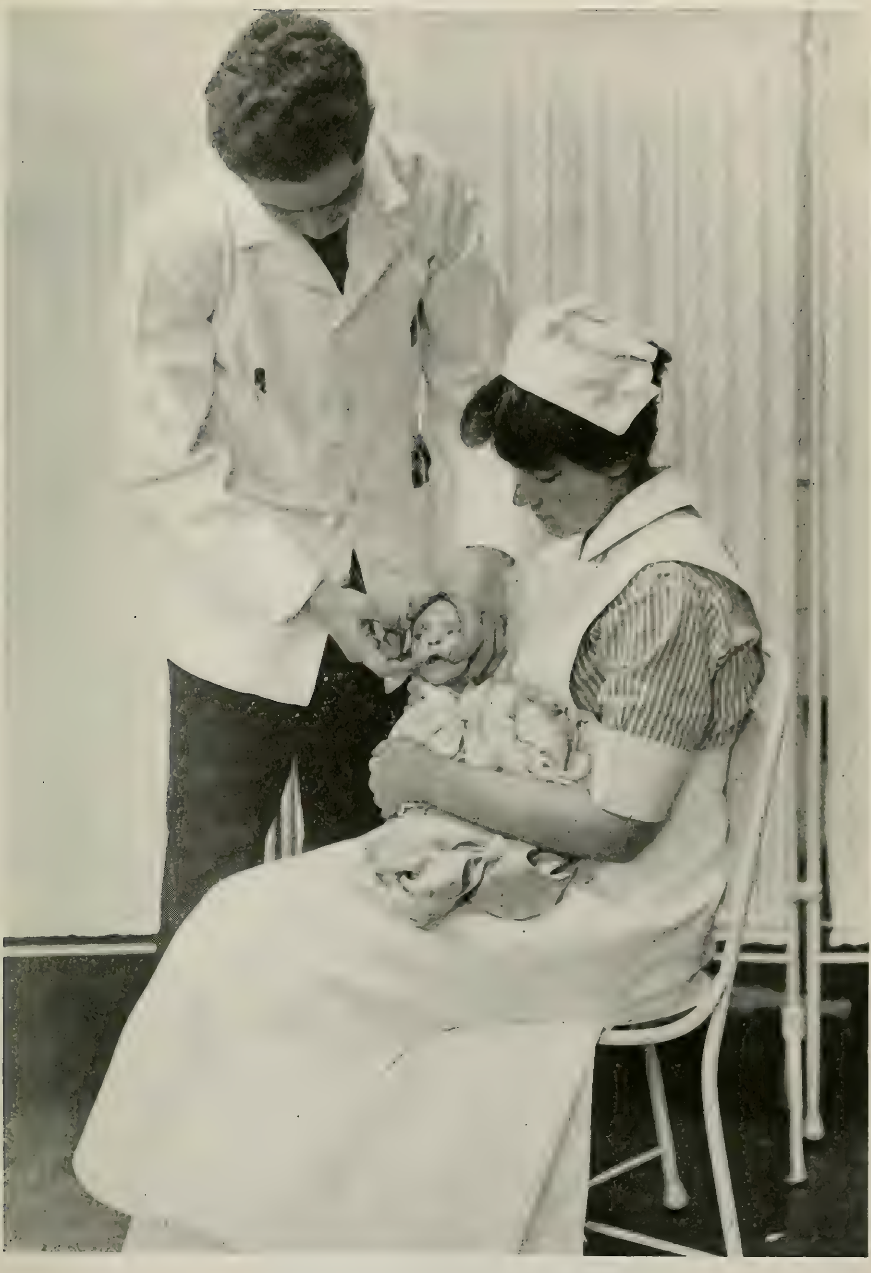


FIG. 235—Position in the examination for adenoids

rise to symptoms. The existence of nocturnal mouth breathing, snoring and a certain amount of disturbance of sleep, without other symptoms, and without impairment of the general health, is not an indication for immediate operation. Proper hygienic management, as described in the prophylaxis of acute rhinitis, or a change of climate, will usually suffice in these cases.

The removal of adenoids is indicated under the following conditions.

1. When obstructive symptoms are marked. If the child is a mouth breather by day as well as at night, or if the adenoid facies is present, or if the disturbance of sleep at night is marked or appears to be influencing the general health, the operation should be recommended.
2. If the child has a chronic nasal discharge, or is subject to frequent colds.
3. When there is any deafness, or a chronic discharging ear, or recurrent attacks of acute otitis media.
4. When the child is subject to enuresis, night terrors, unexplained headaches, stammering, excessive nervous irritability, recurrent attacks of croup, or asthma.
5. When the child is suffering from anemia and malnutrition which cannot be explained in any other way.

CHRONIC HYPERTROPHY OF THE TONSILS

(Chronic Tonsillitis.)

The terms chronic tonsillitis and hypertrophied or enlarged tonsils are commonly used to express the same condition, especially in children, in whom a chronic inflammation of the tonsils unaccompanied by enlargement seldom occurs.

Although the hypertrophy of the faucial tonsils may exist without a corresponding affection of the pharyngeal tonsil, yet it is very apt to be associated with this latter condition.

PATHOLOGICAL ANATOMY.—The enlargement is a true hypertrophy, sometimes accompanied by more or less inflammatory deposit. If the parenchymatous or glandular tissues are especially affected we find a soft, more or less red and vascular tonsil, with large crypts, often containing much secretion. In the interstitial form the tonsil is hard and tough, the crypts less prominent or even very small, and the vascularity much diminished. These types are the two extremes, and are rare; in most cases the enlargement is essentially one of hypertrophy. The tonsils may be only moderately enlarged, or their size may be so increased that they meet, touching each other in the median line.

SYMPTOMS.—The symptoms of hypertrophy of the faucial tonsils vary according to the degree of enlargement. Normally the tonsils can scarcely be seen on inspection of the throat. In examining a child for enlargement of the tonsils care must be taken that the pharynx is not contracted by gagging at the time the examination is made. The act of gagging, which is easily brought about in children by a careless use of the tongue-depressor, brings the tonsils toward the median line, thus giving tonsils of normal size the appearance of being large and obstructive. When only moderately enlarged they may produce no symptoms whatever, or they may be the source of recurrent acute affections of the throat. When considerably enlarged they may still not produce any marked symptoms, provided that the passage of air through the naso-pharynx is unobstructed. They may, however, even when the pharyngeal tonsil is not enlarged, cause obstruction in the naso-pharynx by pressure as they enlarge upward and backward. When this happens, the same interference with the breathing and development of the child takes place as when the obstruction is primarily in the naso-pharynx. These symptoms are the same as have already been described in speaking of hypertrophy of the pharyngeal tonsil, and therefore need not be mentioned in detail here.

Occasionally difficulty in swallowing and thickness of speech may arise when only the faucial tonsils are enlarged.

PROGNOSIS.—The prognosis in cases of hypertrophy of the faucial tonsils depends upon these varied anatomical conditions just described. So long as the tonsils do not encroach on the naso-pharynx the prognosis, so far as injury to the child is concerned, is good. We should remember, however, that the enlargement of the tonsils is a fertile source of irritation which may prepare the way for serious disease produced by the various microörganisms. The prognosis as to their disappearance is not especially good, as when once hypertrophied they seldom recover their normal size without active treatment, though they generally diminish slowly in size after puberty.

TREATMENT.—Local applications for the reduction of hypertrophied tonsils are useless. The most thorough and certain way of curing the disease is by excision. If the pharyngeal tonsil also is hypertrophied the child should have an anesthetic, and the operation for adenoid vegetations and tonsils should be done at the same time. If the tonsils alone are enlarged, they may be excised with the tonsillotome without an anesthetic, provided they are not adherent to the pillars of the fauces. After the operation the child should be made to gargle with a solution of borate of sodium for two or three days and should be given only milk for its food.

CHRONIC FOLLICULAR PHARYNGITIS

In this condition there is a chronic hypertrophy of the lymphoid tissue on the posterior pharyngeal wall. The principal symptom is an irritating cough, which is most pronounced at night and in the morning. On inspection, the enlarged lymph follicles may be easily seen. The mistake should not be made of thinking that these children who are coughing continuously, often losing in weight and looking weak and anemic, are necessarily affected with bronchitis. This series of symptoms is frequently looked upon as diagnostic of bronchitis, when it really arises from pharyngitis, and can be cured by treating the latter disease.

TREATMENT.—The treatment of chronic pharyngitis is to remove any hypertrophic condition of the tonsils, or nose, to regulate carefully the child's general nutrition, and to avoid undue exposure to inclement weather or to air vitiated in any way, as by dust. Local applications of a one per cent solution of nitrate of silver, followed immediately by thorough cleansing with sterilized water, are sometimes indicated in the more intractable cases.

ELONGATED UVULA

Accompanying pharyngitis, usually in its chronic form, an elongation of the uvula is at times met with in children. This condition may arise from a relaxed condition of the muscles of the soft palate and of the uvula, or may consist simply of a redundancy of the mucous membrane at the tip of the uvula. The general irritated condition of the uvula and the tickling sensation produced by its elongated tip touching the base of the tongue cause a harassing cough, which by its persistence weakens the child, gives rise to loss of appetite, and interferes with its nutrition.

A papillomatous growth is sometimes found attached to the tip of the uvula or to its side, which causes the same symptoms as elongation of the uvula. The treatment is excision, after which it does not recur.

TREATMENT.—Local applications of astringents, such as alum, are at times sufficient to restore the uvula to its normal condition, but the disease can be cured quickly by excising the end of the uvula with blunt-pointed scissors. The amputation of the entire uvula is to be avoided, as it has been known to weaken the muscular action of the soft palate.

IV. DISEASES OF THE EAR

OTITIS MEDIA

Infection of the cavity of the middle ear is one of the commonest affections of infancy and early childhood, and is frequently met with in older children.

ETIOLOGY.—A variety of microörganisms have been found in acute otitis media, and in general they are the same varieties as are concerned in the acute infections of the nose, pharynx, larynx, and bronchi. Among them are to be numbered streptococci, staphylococci, pneumococci, and influenza bacilli. At times one of these organisms may be found in pure culture, but mixed infections are very common.

The disease is usually a secondary infection, the primary source being some infectious process in the rhino-pharynx. The route followed by the organisms is through the Eustachian tube.

Otitis media is a particularly common affection following the diseases in which there is involvement of the mucous membrane of the rhino-pharynx. It is consequently often seen in rhinitis, pharyngitis, or tonsillitis, or in children having adenoid vegetations of the vault of the pharynx. For the same reason, it is the most common complication of measles, diphtheria, scarlet fever, and epidemic influenza.

Another factor which strongly predisposes toward the occurrence of middle ear infection, is lowered vitality and resistance. Otitis may follow any of the conditions lowering the resistance, and is frequently seen in pneumonia, whooping-cough, gastro-intestinal disease, congenital syphilis, typhoid fever, and meningitis.

The reasons for the frequency with which otitis media is seen in infancy, are, the low systemic resistance of the infant, its liability both to contagious infections of the upper air passages, and to infections or disturbances of nutrition which still further lower the systemic resistance, and the patulous Eustachian tubes. Adenoids, and the liability to pharyngeal infection caused by adenoids, is the chief etiological factor in older children.

PATHOLOGICAL ANATOMY.—The lesion is an acute inflammation of the mucous membrane lining the middle ear. It is customary to divide the inflammation into two types, catarrhal and purulent. This distinction is based on the character of the exudate, but is of no essential value. The exudate may be serous, seropurulent, or purulent. The symptoms are likely to be more severe with purulent than with serous exudates.

SYMPTOMS.—The only constant symptom of acute otitis media is fever. The temperature ranges from 100° to 104° F. and there is nothing characteristic about the temperature curve.

The only other symptom of importance is pain. In older children when pain is present, definite earache is complained of. In infants and young children, the presence of pain can only be inferred from crying, restlessness, and poor sleep. In rare instances only does the child put its hand to the affected side, or show tenderness to external manipulation. There is usually nothing pointing toward the ear, even when signs of pain are present. In my experience all signs of pain in the otitis media of young children are more often absent than present.

A variety of other symptoms have been described in connection with otitis media, but no one of them is of sufficient constancy to merit enumeration, or to be of aid in the diagnosis.

The onset of acute otitis media is precisely like the onset of any acute infection in a young child, varying in severity. There is a sudden rise of temperature, with more or less constitutional disturbance, restlessness, loss of appetite, and occasionally vomiting. The children often look quite sick.

When a child develops otitis media, it is often suffering from some primary disease, the symptoms of which are all that are noted. If fever is already present, there is often nothing to mark the involvement of the middle ear. If fever is not already present, the involvement of the ear is marked by a rise of temperature, which cannot be accounted for by the course of the primary infection. Many cases occur apparently as primary infections in infants. If there is an inflammatory process in the rhino-pharynx, it is not sufficient to cause symptoms. In these cases the physician may be puzzled as to the diagnosis, unless he remembers the peculiarities of otitis media in children.

On examination of the ear, there is in the early stage merely marked redness and congestion of the membrana tympani. Later there is distinct bulging. Still later, a perforation may be seen.

COURSE.—In some cases congestion only may be present, and may subside without the formation of exudate. Occasionally a serous exudate will absorb spontaneously, but it will usually become purulent if not allowed to escape. In most cases, spontaneous perforation of the ear drum will eventually occur, if the membrane is not incised. Perforation may occur within twenty-four hours, or not until after a number of weeks; it usually occurs in about a week or ten days. After either spontaneous perforation, or paracentesis, the discharge usually persists for one or two weeks. The ear drum tends to heal very rapidly, and must often be kept open, to prevent the exudate from reaccumulating. Finally, the discharge ceases, and the drum closes.

In a certain number of cases, the disease becomes chronic. This condition is commoner in older children than in infants. The fever subsides, but a large perforation persists in the membranum tympani, and there is a persistent discharge from the ear. Deafness gradually develops. *A certain proportion of cases of chronic otitis media are due to tuberculous infection.*

DIAGNOSIS.—The disgnosis is easily made by otoscopic examination, with which every physician having practice among children must be thoroughly familiar. Nevertheless, otitis media in early life is very frequently overlooked, because the possibility of middle ear disease is not suspected, and the ears are not examined. One reason for the overlooking of otitis media is that definite symptoms pointing toward the ear are wanting. Many text-books have taken great pains to enumerate and emphasize the various symptoms, laying stress on the symptom of pain. Both pain, and localizing symptoms are so frequently absent, that the physician should never depend upon their presence in suspecting otitis.

The ears should be frequently examined as a routine measure in measles, scarlet fever, diphtheria, epidemic influenza, and pneumonia. In all other infectious diseases, whenever the temperature behaves in an unusual manner, failing to fall at the usual time, or rising suddenly without obvious cause, the ears should be examined. In any young child showing the symptoms of an acute infectious disease, without the nature of the infection being immediately obvious, or in any young child having a sudden rise of temperature, the possibility of otitis media should be the first thing to come to the mind of the physician.

COMPLICATIONS AND SEQUELAE.—In comparison with the frequency of otitis media, complications are rare. Most of them are, however, of a serious character.

MASTOIDITIS.—This is the most frequent complication. It is caused by extension of the infection into the mastoid cells, causing suppuration and necrosis of those tissues. In infancy the mastoid cells have not developed, the only cavity in the small mastoid process being the antrum. The mastoid antrum is probably involved in most of the severe or prolonged cases of otitis media, the pus being discharged into the middle ear. Mastoiditis is only seen when the mastoid cells have developed, and is consequently not often seen in the first year of life, and is comparatively rare in the second year. It should be watched for carefully in every case of otitis media in a child over two years.

The most important symptoms are fever, mastoid tenderness, and swelling. The fever is of a septic type, irregular, often reaching a high point, 104°-105° F., at some time in the twenty-four hours, and often falling below normal at another time. Mastoiditis should be suspected when in the course of a case of acute or chronic otitis media a rise of temperature or an increase in constitutional disturbance occurs, which cannot be explained either by the local appearances seen in the ear, or by any evidences of infection elsewhere. In such cases the otoscopic examination of the ear may suggest that the opening in the drum is large enough to permit free drainage, and that the process in the middle ear is improving. Mastoiditis should also be suspected when spontaneous rupture of the drum, or paracentesis, with a free discharge of pus, is not followed by a fall of temperature, but a septic temperature continues.

The suspicion of mastoiditis is confirmed by the finding of tenderness over the mastoid. A slight amount of tenderness is often difficult to detect; it is usually found, not at the tip of the mastoid, but higher up, just behind the auricle. With the tenderness there if often redness and swelling of the tissues over the mastoid.

Mastoiditis, if not operated upon, may terminate in the formation of a post-auricular abscess, due to spontaneous rupture through the bony wall. There is always danger that mastoiditis may give rise to a general septicemia.

MENINGITIS.—This complication, while comparatively rare, is of sufficient importance to be borne in mind as a possibility. It is caused by the extension of infection to the membranes of the brain, either through the roof of the tympanum, or, more rarely, through the internal auditory meatus. It may occur in either acute or chronic cases of otitis media. The most frequent organisms causing the meningitis are the streptococcus and the pneumococcus. The meningitis may be localized, a pachymeningitis, with the formation of pus outside the dura, or there may be a generalized leptomeningitis. The symptoms are those of an acute meningitis. In my experience the onset of acute meningitis originating from the middle ear is apt to be more insidious, and the meningeal picture less clear, than in cases of meningitis of hematogenous origin. After the meningeal symptoms are developed, the course is rapid, and the termination almost invariably fatal.

CEREBRAL ABSCESS.—This complication, also rare, is due to direct extension of the infection through the dura to the brain. The abscess is most frequently seen in the temporal lobe. Abscesses may occur as the sole complication, or may be accompanied by meningitis or sinus thrombosis. Cerebral abscesses are usually seen in old chronic cases of otitis.

THROMBOSIS OF THE LATERAL SINUS.—The sinus may be the seat of a fibrinous clot without infection, or the thrombus may become infected by extension. In the former condition, there are no symptoms sufficiently constant or characteristic to permit a clinical diagnosis. I have seen at autopsy associated with sinus thrombosis, pulmonary infarction, which during life was mistaken for bronchopneumonia.

Septic sinus thrombosis usually occurs as a complication of mastoiditis, and often follows a mastoid operation. The symptoms are severe and suggest a septicemia, and indeed, the secondary lesions of pyemia may be present. There is at first septic fever, then marked prostration, with the sudden development of cerebral symptoms and death. Meningitis may be present. Septic sinus thrombosis should be suspected whenever septicemic or meningeal symptoms develop after a mastoid operation. The diagnosis can only be positively confirmed by blood cultures.

SUPPURATION OF THE LABYRINTH.—This complication is rare. It is most frequently seen in severe cases of otitis media complicating scarlet fever in very young children. It results in complete deafness.

FACIAL PARALYSIS.—This is due to the extension of the inflammation to the canal in the petrous portion of the temporal bone, through which the facial nerve passes. The paralysis is like the common peripheral facial paralysis due to neuritis, all the segments of the nerve being usually involved, and the patient unable to close the eye.

TREATMENT.—At the onset of an attack of otitis media, free catharsis should be employed. In this early stage, examination of the drum membrane will usually show congestion only, but no bulging. The ear should be syringed every two hours, with either a saturated solution of boric acid, or normal saline solution, at a temperature of 110° F. Sodium bromide, in doses of two grains every four hours to a child of two years should be given. I believe that bromide has a most favorable influence, both on the restlessness and discomfort of the child, and on the inflammation. Under this treatment, in a certain number of cases, the congestion of the drum will subside and the temperature will fall.

It is not wise to allow otitis media to exist more than forty-eight hours without operation. If, after twenty-four to forty-eight hours, the temperature has not fallen, and the evidences of pain have not subsided, or if within this period, the temperature has become higher and the evidences of pain have increased, operation should be considered. The most reliable guide is neither temperature, nor

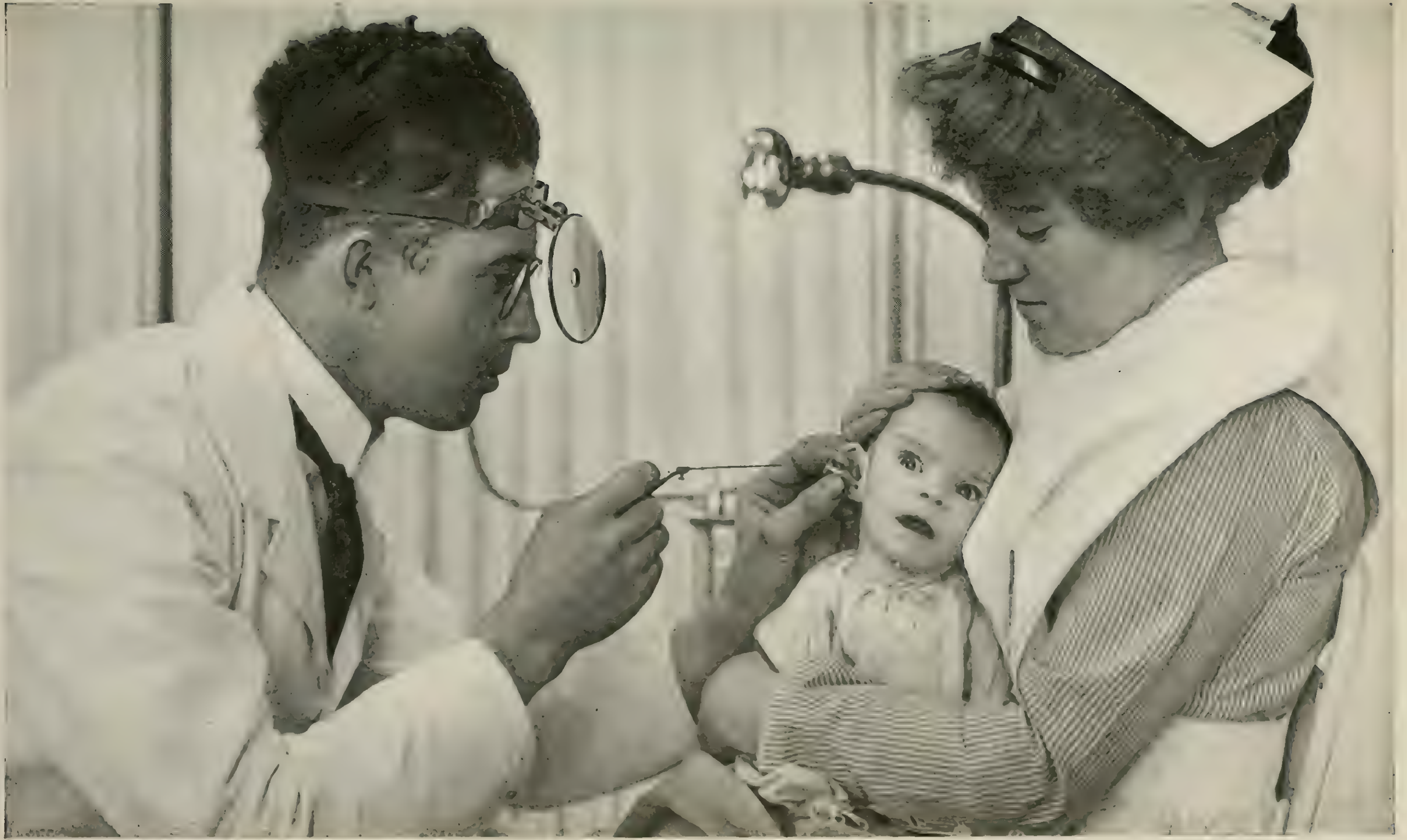


Fig. 236—Paracentesis of the membranum tympani

symptoms, but the appearance of the drum membrane. If the congestion of the membrane does not subside, or at any time if there is any bulging of the membrane, paracentesis should be performed. The presence of mastoid tenderness is always an indication for immediate incision of the drum membrane.

If bulging is present, the incision should be made at the most prominent point; otherwise, it should be made in the lower posterior quadrant, extending upward through Shrapnell's membrane. In cases incised for persistence of congestion or symptoms, the discharge will usually consist only of blood, but nevertheless marked relief will be afforded. In early cases with fluid, the discharge will usually be serous, while in later cases it will be purulent.

After incision, the syringing with hot normal saline, or saturated boric acid solution should be continued at two to four hour intervals according to the amount of discharge. In the majority of cases the discharge will cease in from one to three weeks. A serous discharge usually ceases within ten days, while a purulent discharge is more persistent. If the discharge ceases abruptly, the usual cause is closure of the opening in the drum, either by thick pus, or by premature healing of the membrane. This may lead to reaccumulation of exudate, and the membrane should be reopened. If the discharge persists longer than the usual period, the usual causes are either adenoids, or poor general health and resistance. If a persistent discharge has a fetid odor, peroxide of hydrogen, one part to two of water, should be used in the syringing.

In older children especially, *earache* is sometimes severe in the early stages of otitis media, and attacks of severe earache are seen in cases which do not go on to the formation of exudate. In these cases, instead of simple syringing, irrigations with a pint of hot saline solution should be given with a fountain syringe, at an elevation of two feet. Between the irrigations the child should lie on a hot water bottle. Neither warm olive oil, nor laudanum should be dropped into the ear. A few cases are severe enough not to be relieved by hot irrigations and bromide, and require an opiate. Cases, however, in which earache persists, require paracentesis.

Fully developed cases of mastoiditis require surgical operation. It must be remembered, however, that the only effective operation is a radical one, and is decidedly serious. It is attended with considerable immediate risk, and I have seen a number of cases in which death occurred either immediately after the operation, or later from some intercurrent infection. I believe also that the dangerous complications supposed to follow delay in operating, such as septic sinus thrombosis, meningitis, and cerebral abscess, are not so common as the surgeons seem to think they are. The operation should certainly not be performed upon the mere suspicion of mastoid involvement. In the early stage, when there is often some doubt as to whether there is actually tenderness over the mastoid, free drainage of the middle ear should be established, an ice bag should be applied continuously to the mastoid, and blood may be withdrawn by means of the artificial leech. With these measures, the symptoms pointing toward mastoid involvment often subside. The operation should only be undertaken when clear and positive indications are present. Tenderness over the mastoid is difficult to determine, especially in young children. The physician should be sure that the septic temperature is due to no other discoverable cause, and that mastoid tenderness is really present. With clear indications, the operation should be performed, if the symptoms do not subside within twenty-four hours.

DIVISION XI

DISEASES OF THE LIVER, PANCREAS, AND PERITONEUM

I. DISEASES OF THE LIVER

GENERAL ETIOLOGY AND PATHOLOGICAL ANATOMY.—The difficulties in presenting a scientific classification of the diseases of the liver which will be of any clinical utility are almost insuperable. The liver is a large parenchymatous organ of highly specialized structure, with a very rich blood supply containing the products of absorption from the gastro-intestinal canal. One would naturally suspect that such an organ would be particularly susceptible to injurious influences, and that these influences would be likely to produce not only disturbance of function but also actual anatomical lesions. This is indeed the case. A variety of very definite hepatic lesions have been described, and there has been shown to exist a more or less close correspondence between the nature of the injurious influence, and the type of tissue lesion produced. There is, however, a much less close correspondence between the lesions of the liver, and the clinical manifestations of disease. Many of the hepatic lesions, especially those found in early life, are unaccompanied by clinical manifestations. Furthermore, several types of lesion may produce practically the same clinical picture. Again some of the lesions found in the liver are very rare in early life, but still have been reported often enough to merit enumeration, if not detailed description.

Many of the pathological conditions found in the liver are *secondary*, that is, are part of the manifestations of disease involving other parts of the body.

MALFORMATIONS.—Congenital malformations and malpositions may occur. The most important congenital disease of the liver is *congenital cirrhosis and obliteration of the bile ducts;* this has been already described under the Diseases of the Newborn. Other malformations are too rare to be of clinical interest.

MECHANICAL INJURIES.—The liver in childhood is not injured by external trauma, except under the most exceptional circumstances. There is a form of cirrhosis associated with hemochromatosis, in which the action of the pigment particles on the liver is purely mechanical. I have not heard of a case of this kind recognized in childhood. The *passive congestion* of the liver, which occurs in cardiac disease, is a mechanical process in-so-far as the liver is concerned. It occurs in children from the same causes as in adults, namely, congenital or acquired disease of the heart, pulmonary atelectasis, chronic pleurisy, emphysema, and chronic interstitial pneumonia. There are no characteristic clinical manifestations, except moderate enlargement of the liver, often with tenderness, and sometimes with pain.

NEW GROWTHS.—Tumors of the liver are rare in early life. The usual form of new growth is *sarcoma*. The involvement of the liver is usually secondary, the tumor originating elsewhere, most often in the adrenal tissue of the kidney. Primary sarcoma of the liver, however, has been observed. There have been rare instances of hydatid cysts of the liver in children.

The clinical picture in sarcoma of the liver is that of a slowly increasing enlargement of the organ with progressive asthenia. The diagnosis depends on the recognition of the primary site of the new growth.

FUNCTIONAL DISTURBANCES.—Disturbance of hepatic function without demonstrable anatomical lesions is a rare condition in children. The various causes which in children so often produce disturbance of function, particularly in the digestive and nervous systems, produce in the liver either definite anatomical lesions, or else no recognizable disturbance. *Gall stones* have been observed in children, but are rare.

TOXIC LESIONS.—The most common lesions found post-mortem in the livers of infants and children are of toxic origin. The toxins may come from a great number of sources. Toxic substances such as *alcohol* may be introduced into the intestinal canal through the mouth, and after absorption are carried directly to the liver by the portal vein, acting upon the liver in such a way as to produce characteristic lesions. It is in this way that *alcoholic cirrhosis* is produced. Toxic substances may be formed in the intestinal canal as a result of bacterial fermentation or of abnormal chemical changes in the food, and these also may produce lesions. Again, toxic substances may be present in the general circulation. Their origin may be either some general or localized infection, the toxins being the product of bacterial activity, or it may be some disturbance of metabolism which causes the formation of toxic products.

The lesions of the liver tissue produced by toxins are various. The most common and typical is *central necrosis*, in which a necrosis of the liver cells, most marked about the hepatic veins, occurs. This is an acute and often terminal condition, seen most often in cases which have died from infection or general toxemia. In these cases there are no clinical manifestations corresponding to the lesion of the liver. There is an extreme type of this lesion, which does have clinical manifestations, and which has long been recognized under the name of *acute yellow atrophy.* This disease occurs most often in young adults, but occasional cases have been reported in early life. The cause is not definitely known; it is probably some unusually severe toxemia, and it is probable that more than one variety of toxemia can produce the hepatic lesions characteristic of the disease. The symptoms and course of the disease as reported in early life are the same as in adults, the characteristic features being intense jaundice with marked asthenia, advancing progressively to a fatal ending.

In infants who die of chronic gastro-intestinal disease, there is usually found at autopsy a more or less extensive necrosis of the cells of the hepatic parenchyma most marked about the portal veins. This lesion probably represents the action of toxin absorbed from the intestinal canal. Another condition frequently found in the liver in infants, particularly in those dying of chronic diseases, is *fatty infiltration.* This is often associated with the necrosis just mentioned. More than half the cases coming to autopsy at the Infant's Hospital have fatty livers. This condition is characterized by a complete absence of all clinical manifestations.

Amyloid degeneration of the liver is comparatively common in early life, and is probably of toxic origin. As this lesion is recognizable clinically, it will be described further in the following pages.

INFECTIONS.—The liver, like all other organs, is liable to invasion by pathogenic microörganisms. Some of the infections of the liver represent only a part of a more or less generalized infectious process. In tuberculosis the infection may extend to the liver, either in the form of miliary tuberculosis or of solitary tubercle, but these lesions have no characteristic symptoms. The infections of the liver of clinical importance are *catarrhal jaundice* and *suppurative hepatitis.*

CATARRHAL JAUNDICE

This is the commonest disease of the liver encountered in early life. It is rare in infancy, and is almost unknown in the newborn. It is quite common throughout childhood after the third year.

PATHOLOGICAL ANATOMY.—The jaundice is obstructive in type, and the obstruction is due to catarrhal inflammation and swelling of the mucous membrane lining the bile ducts. Owing to the symptoms of indigestion which accompany catarrhal jaundice, it was at one time held by many authorities that it is the mucous membrane of the duodenum which is chiefly involved in the catarrhal swelling, causing obstruction at the opening of the common bile duct. This view was the origin of the term *gastro-duodenitis*, which has been widely employed as a synonym for catarrhal jaundice. It has, however, since been recognized that the principal site of the lesion is not the mucous membrane of the gut, but the mucous membrane of the bile ducts themselves. Moreover, there is evidence that it is not only the mucous membrane lining the *common* bile duct which is involved in the process, but also the mucous membrane of the bile ducts throughout the liver. One important evidence that catarrhal jaundice represents not merely an obstructive condition of the common bile duct, but rather a widespread disease involving the whole liver and leading to pronounced disturbance of the function of the organ, is found in the results of certain modern physiological tests of hepatic function. The galactose test, based upon the ability of the liver to store sugar, shows a practically constant diminution of functional sufficiency in catarrhal jaundice. This would indicate that the cause of catarrhal jaundice, in addition to producing biliary obstruction, produces functional disturbance of the hepatic parenchyma.

ETIOLOGY.—At one time the view was held that the cause of catarrhal jaundice is a functional disturbance of digestion. Under this view the irritation of the products of digestive disturbance was supposed to produce catarrhal inflammation of the duodenal mucous membrane, which might extend into the common bile duct. This was the view of Rotch, who described catarrhal jaundice under the name of *acute duodenal indigestion*. There has never been any scientific evidence in support of this theory of the etiology of the disease.

More modern opinion is almost unanimous in regarding catarrhal jaundice as representing an *infection*. There is some difference of opinion as to whether the infectious process originates in the duodenum as a primary duodenitis, and spreads to the bile ducts, or whether the bile ducts are primarily attacked. There is, of course, no doubt that the microörganisms gain access to the bile ducts from the duodenum. The fact that catarrhal jaundice is rare in infancy, the period when intestinal infections are most common, and that it is practically never seen as a complication of the recognized forms of intestinal infection, points to the probable primary character of the process in the bile ducts. I believe that this is the most probable view.

The nature of the microörganisms which produce the catarrhal inflammation of the bile ducts is as yet unknown. Various organisms have been described as associated with the lesions. A very plausible theory is that the process is due to a mixed infection with organisms of comparatively low virulence, and that etiologically it corresponds to the catarrhal infections of the upper air passages, which are described as rhinitis, pharyngitis, laryngitis, and bronchitis. There is a possibility, however, that the process may be more specific. The disease often occurs in *epidemics*, and a number of widespread epidemics have been described, especially in Europe. While this does not necessarily suggest a specific organism as yet unknown, it raises interesting questions in connection with certain recent work on the selective affinities of bacteria. This will be further discussed under problems and research.

If the prevalent view of the infectious nature of catarrhal jaundice be accepted, I believe that a great improvement can be made in the descriptive terminology of the disease. From the scientific point of view the term "catarrhal jaundice" is very unsatisfactory. The term "catarrhal," while originally based on an ancient and wholly false theory of disease, and while subject to much abuse in the literature of quack medical advertising, is nevertheless very useful in describing a certain form of inflammation of mucous membranes. No satisfactory substitute for "catarrhal" as used in this sense has yet been found. The term "jaundice," however, is objectionable as the name of a disease. It is only the name of a symptom which is seen in many diseased processes, and conveys nothing either of etiology or of pathological anatomy. The title "catarrhal jaundice" has been used in this book only on account of its general employment by the medical profession. If the essential feature of the disease is an inflammation of the bile ducts, it is a *cholangitis*. The writer begs to suggest the term *catarrhal cholangitis* as far preferable to catarrhal jaundice, and as adequately descriptive of the pathological anatomy and probable etiology of the disease.

SYMPTOMS.—The onset of the disease is acute and resembles an attack of acute indigestion, with vomiting and abdominal pain. There is fever, which is usually slight, but occasionally may be marked, especially at the onset. There is a moderate amount of prostration. Occasionally older children complain of chilly sensations at the beginning of the attack. The abdominal pain may be quite severe, and may be associated with tenderness under the liver.

After two or three days, *icterus*, the characteristic symptom of the disease, appears. It is seen first in the conjunctivae, and then in the skin, varying in intensity with the severity of the attack. It is usually not very intense, and never shows the marked greenish-yellow color which is so often seen in congenital obliteration of the bile ducts. The other signs of obstructive jaundice are present. The stools are gray or white, and contain no bile pigment. The urine is very dark, often of a yellowish-green color, and stains the clothing. The test for bile is strongly positive.

During this stage of the disease, the patient is very dull and languid, and often complains of headache or of general discomfort. Anorexia is very marked. The tongue shows a heavy white coat. The bowels are constipated, there is usually flatulence, and there may be abdominal distention. The liver is often but not always found slightly enlarged, and may be tender to pressure. There is occasionally enlargement of the spleen. The slow pulse and itching skin so common in adults are not common, but are occasionally seen in older children. There is sometimes albuminuria, the urine containing also renal epithelium and casts.

The course of the disease is usually short. After a few days the temperature falls to the normal. The general symptoms then disappear, the icterus persisting somewhat longer. The usual duration of the disease is from one to two weeks, but in exceptional cases more prolonged attacks, and also recurrences, are seen.

DIAGNOSIS.—Usually the diagnosis is extremely easy. Except in the newborn when the disease is very rare, obstructive jaundice is due to no other cause. The few cases of acute yellow atrophy of the liver occurring in childhood have usually been first diagnosed as catarrhal jaundice. Until the progressive asthenia of this condition begins to suggest an approaching fatal ending, differentiation is impossible, but this disease is too rare in early life to enter into consideration as more than a remote possibility. In the newborn, catarrhal jaundice is a possibility, though extremely rare. It can only be differentiated from congenital obliteration of the bile ducts by its favorable course.

PROGNOSIS.—The prognosis is invariably favorable. The only unfavorable possibility is the remote one of the disease being acute yellow atrophy.

TREATMENT.—The treatment is mainly dietetic. The absence of bile in the duodenum indicates that the fats and carbohydrates should be excluded, or reduced to a minimum. During the first day, when vomiting is often marked, nothing but water should be given. Later, skimmed milk diluted with lime water, or mutton or chicken broth, is given in small quantities, if the child asks for food. He should not be urged to eat. When the child becomes hungry, rare meat, and a little toast may be added. This diet of skimmed milk, broths, meat, and toast, in small quantities, should be continued until the jaundice has disappeared. The green vegetables are next added, then fruit, then the cereals and other starchy foods, and finally the fats. Water may be given freely. If vomiting tends to continue, the alkaline mineral waters are often better borne.

No medication is usually needed, except to keep the bowels open. Citrate of magnesia, Carlsbad salts, or rhubarb and soda, are useful for this purpose. Calomel should not be given. Occasionally pain is severe enough to require counterirritation. If the hot water bag fails to give relief, the mustard paste should be tried, and if this is not effective, the turpentine stupe should be used.

Children with catarrhal jaundice should be kept in bed as long as they have any fever, and also until they are actively desirous of getting up.

PROBLEMS AND RESEARCH.—In catarrhal jaundice the principal problem is that of etiology. Positive proof that the disease represents an infection is still lacking, although, as stated under etiology, all the evidence points in that direction.

The nature of the infection is the most important problem. It was stated under etiology that while the theory of a mixed infection with non-specific organisms of low virulence is a plausible one, there is some evidence that the infection has more specific characteristics. The recent epoch-making work of Rosenow on the transmutation of bacteria, which has already been referred to many times in this book, is of marked interest in connection with catarrhal jaundice, suggesting a future solution of the problem. Rosenow's work has apparently proved that members of the streptococcus-pneumococcus group can undergo transmutation in variations of morphology, cultural characteristics, biological reactions, and general and special pathogenicity. Still more important is his apparent proof that bacteria can acquire elective tissue affinity in foci of infection as well as through cultivation and animal passage. Strains of streptococci having an acquired elective affinity for the gall bladder have been demonstrated. Is it not possible that catarrhal jaundice may be produced by microörganisms having a similar elective affinity for the bile ducts, and that the reported epidemic occurrence of the disease may be explained in this way? Under this theory the occurrence of sporadic cases would be explained by the acquirement of elective tissue affinity in some primary focus in the mouth, tonsils, or intestinal canal.

The study of hepatic function by means of modern physiological tests has produced a voluminous literature in the last few years. It has been demonstrated that the galactose test shows a diminution in hepatic function in catarrhal jaundice. The phenoltetrachlorphthalein test is not adapted to the study of this disease. In general, such tests have no especial clinical utility in children. The diagnosis of catarrhal jaundice is easy in early life, and the various other conditions in which the tests are useful in adults, do not occur.

SUPPURATIVE HEPATITIS

(Abscess of the Liver)

Abscess of the liver is an exceedingly rare condition in both infants and children. Even when septic infection of the umbilical vessels has taken place at birth, it has been found that only in a very small proportion of these cases has hepatic abscess resulted.

ETIOLOGY.—According to Musser's analysis of thirty-four reported cases, the average age was nine years, and the youngest one year. The causes in these cases were from traumatism, from roundworms entering the bile ducts, from pylephlebitis in four cases, from umbilical phlebitis once, from pyemia twice, and once each from pelvic peritonitis, dysentery, perityphlitis, malaria, and tuberculosis of the lungs. Musser therefore concludes that the general conditions, such as climate and habits of life, which are marked etiological factors in adults, are not of significance in children.

SYMPTOMS.—The onset, the progress, and the duration of the disease depend greatly on the primary cause. Icterus may occur, but usually is not marked, and is not of much significance. The local symptoms connected with the liver are the same as in adult life, and are chiefly pain, not always, however, in the hepatic region, but in different parts of the abdomen, enlargement of the liver, usually downward, and tenderness over the liver. If in addition to these symptoms a tumor is detected apparently connected with the liver, and accompanied by fever of a hectic, pyemic, or intermittent type, with sometimes an initial chill or with irregular daily chills, abscess of the liver is to be strongly suspected. Leukocytosis is present. The symptoms are at times, however, very latent, and the diagnosis can only be determined by aspiration.

The duration of the disease is apt to be prolonged, and the child gradually emaciates and fails in strength.

The prognosis varies greatly, as there are so many conditions depending upon the especial cause and the especial locality of the abscess which must be taken into account and which render the chances of recovery more or less uncertain.

TREATMENT.—The treatment is essentially surgical. Musser reports twelve recoveries out of the thirty-four cases, and in eleven of these aspiration or incision or both were employed.

AMYLOID DEGENERATION

This lesion occurs in the liver in children as well as in adults.

ETIOLOGY.—Amyloid degeneration is particularly associated with chronic infectious processes in other parts of the body. The changes in the liver are associated with a similar pathological process in other organs, particularly in the spleen and kidneys, and sometimes in the villi of the small intestines. It is probable that this form of degeneration is due to the prolonged action of bacterial products. It has been shown experimentally by Krakow, Davidsohn and others, that amyloid degeneration can be produced by prolonged infection with the staphylococcus aureus. The condition is particularly associated with infections involving prolonged suppuration, such as osteomyelitis, tuberculous bone and joint disease, and empyema. It is also seen in syphilis, and in chronic tuberculosis without bone lesions. It is probable therefore, though not experimentally proved, that amyloid degeneration can be produced by the products of various forms of bacteria.

PATHOLOGICAL ANATOMY.—Macroscopically the liver is very much enlarged, its substance is very firm and hard, and its appearance is waxy and glistening. The application to the surface of a solution of iodin gives a characteristic mahogany color. Microscopically, the amyloid substance is seen to be deposited mainly between the capillaries and the liver cells. This causes occlusion of the vessels and atrophy of the cells as a result of pressure.

SYMPTOMS.—The amyloid infiltration in itself produces few symptoms, the clinical picture being mainly determined by the primary disease. There are no clinical evidences of disturbed hepatic function, such as icterus. Edema of the extremities and ascites are rare, and when they occur, they are due to associated renal disease, or to pressure of the enlarged liver upon the vena cava.

The most characteristic general symptom is the very marked secondary anemia which develops in the course of the disease. Children with amyloid degeneration show a marked cachexia, with a peculiar waxy pallor, seen in no other condition, and suggestive of malignant disease.

The liver is usually very much enlarged. The edge may reach the umbilicus, or even lower. The surface feels smooth and hard, and the edge smooth and rounded. There is no tenderness. The spleen is always enlarged to a varying degree. Albuminuria, with waxy casts, and edema and ascites, may be present as a result of the associated amyloid infiltration of the kidneys.

The disease is slowly progressive, running a long course, often of years. The primary cause is often not removable, and in such cases the progress is uninterrupted. In cases in which the primary cause is eliminated, the process in the liver may remain stationary for a long time. Death occurs eventually, either from exhaustion, nephritis, or intercurrent disease.

DIAGNOSIS.—Amyloid infiltration of the liver must be distinguished from other conditions in which the liver is enlarged. These are *passive congestion*, *cirrhosis*, *abscess*, *sarcoma*, *syphilis*, *fatty, infiltration*, and *leukemia*.

In *passive congestion* the liver is usually tender as well as enlarged. The condition is mainly recognized by the clinical evidences of the disease of the heart or lungs which causes the congestion of the liver. The liver is enlarged in certain stages of *cirrhosis*, but this is a rare condition in early life. Ascites is usually present. *Abscess* is excluded by the absence in amyloid of chills, fever, and tenderness, and by the very chronic course of the latter. *Sarcoma* is recognized by the finding of a primary sarcoma elsewhere. An irregular or nodular surface of the liver is against amyloid. *Syphilis* of the liver with enlargement occurs mainly in young infants. If in a case of syphilis the liver is found very large and hard in later childhood, the process is probably amyloid. *Fatty infiltration* rarely produces an enlargement of the liver marked enough to be mistaken for amyloid. *Leukemia* is recognized by the characteristic changes in the blood.

The diagnosis of amyloid, and its differentiation from other causes of enlarged liver, usually rests mainly on the finding of definite evidences of some prolonged suppuration or other chronic infection. The waxy cachexia, albuminuria, and very chronic course, are aids in the recognition of the condition.

PROGNOSIS.—The prognosis is unfavorable. A few cases of improvement, or even of cure have been reported from treatment of the primary cause. Most cases eventually die.

TREATMENT.—The treatment is that of the primary chronic infection. Syphilis should be actively treated. Chronic suppuration should receive proper surgical attention. Chronic tuberculosis should receive outdoor treatment.

CIRRHOSIS OF THE LIVER

(Interstitial Hepatitis)

Cirrhosis of the liver is a condition characterized by proliferation of the interstitial tissue of the liver, with the formation of fibrous tissue.

ETIOLOGY.—The only common cause of cirrhosis of the liver in infancy is *syphilis*. Other forms of cirrhosis are very rare in early life; they are seen most frequently in late childhood, between the ages of nine and fifteen years. In the majority of these cases the etiology is obscure. The lesion most often resembles that seen in alcoholic cirrhosis, but *alcoholism* was found in only fifteen per cent of Howard's collection of sixty-five cases. Syphilis and tuberculosis were present each in eleven per cent. In the cases of obscure etiology, the lesion usually resembles that of alcoholic cirrhosis, and the most plausible theory is that the cause is some *toxin* formed in the intestinal canal. *Tuberculous peritonitis* is the form of tuberculosis with which cirrhosis of the liver is sometimes associated. The association of hepatic cirrhosis with chronic *adhesive pericarditis* was mentioned in the description of the latter disease. The cirrhosis has been attributed to the marked chronic passive congestion produced in the liver by pericardial adhesions, but the connection between the pericardial and hepatic lesions is not quite clear. Finally, cirrhosis of the liver has been observed to follow acute general *infections* such as measles and scarlet fever, and there is no doubt that in some cases the lesions of cirrhosis are due to the direct presence of bacteria, which probably gain entrance through the bile ducts.

PATHOLOGICAL ANATOMY.—In most forms of cirrhosis there is necrosis or other degenerative change in the liver cells. These degenerative changes may be produced by various injurious influences acting on the liver, mainly toxins and infections. Mallory has shown that when the injurious influence acts only on the liver cells, there is no proliferation of the interstitial tissue. In cirrhosis there must be injury to the fibroblasts. Mallory has also shown that there is a correspondence between the cause and the tissue reaction. In syphilitic cirrhosis injury to the liver cells is less marked than in other forms, but there are immense numbers of treponemata in the liver, and a very marked increase in fibrous tissue and in the size of the organ. In alcoholic cirrhosis, and in the types seen in children with an obscure etiology, there is marked hyaline degeneration of the liver cells beginning near the portal vessels, marked fatty infiltration, and active regeneration of connective tissue. The liver becomes first enlarged, later contracted. In infectious cirrhosis there is extensive inflammatory reaction and marked increase of fibrous tissue, extending from the periphery of the lobule toward the hepatic vein. The liver is markedly increased in size. The so-called Hanot's hypertrophic biliary cirrhosis with jaundice is extremely rare in early life.

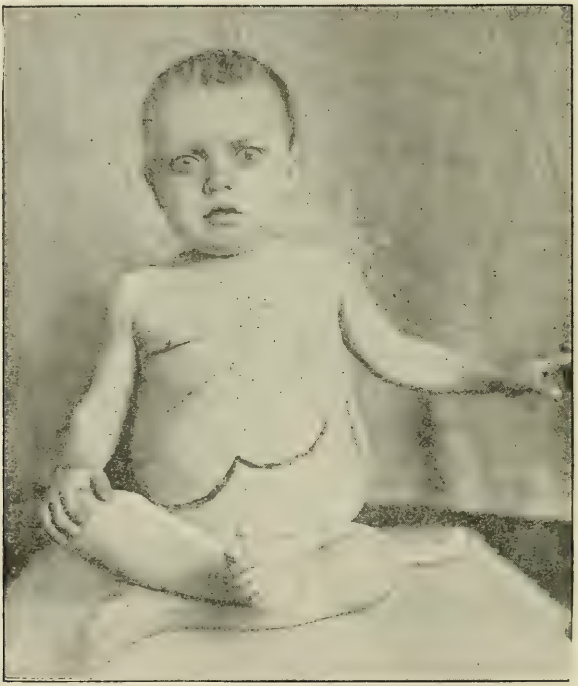
SYMPTOMS.—In the syphilitic cirrhosis of infancy, there are no symptoms particularly caused by the hepatic lesion. The only manifestation is the marked enlargement of the liver.

In the cases of cirrhosis occurring most often in later childhood, the symptoms are the same as in adult life. In the early stages there are indefinite symptoms suggesting disturbance of digestion, such as occasional attacks of nausea and vomiting from relatively slight cause. These symptoms are usually so little marked that they do not attract attention. Examination in this stage may show slight enlargement of the liver.

With the advance of the disease process, the most characteristic symptom is *ascites*, which is usually moderate in amount at this stage. The liver may be enlarged, or may be of normal size. The surface may seem somewhat irregular to palpation. Next to ascites, the most characteristic symptom of this stage is dilatation of the superficial veins of the abdominal wall. The spleen is usually enlarged. There may be hemorrhages from the nose, stomach or intestines. Icterus may be present, but is usually slight.

In the late stage of the disease ascites is very marked, and occasionally there is general dropsy. The liver is smaller than normal and is usually not palpable. Diarrhea is often present. Cachexia becomes very marked, the course of the disease is more rapid than in adults, and death occurs either from exhaustion, or from a terminal general streptococcus infection.

FIG. 237



Hypertrophic cirrhois. Female, 18 months old

DIAGNOSIS.—In early infancy a marked enlargement of the liver usually means syphilitic cirrhosis. In late infancy and childhood, if enlargement of the liver is unaccompanied by ascites, cirrhosis should be considered as no more than a very remote possibility. All the other causes of enlargement of the liver, namely, passive congestion, abscess, sarcoma, fatty infiltration and leukemia, are more common than cirrhosis as a cause of hepatic enlargement. Only when all these conditions can be excluded, should cirrhosis be suspected.

A more difficult problem in diagnosis is presented when ascites is the principal clinical manifestation. The other conditions causing ascites in children are *cardiac disease*, *nephritis* and *tuberculous peritonitis*. It must be remembered that all these are much more common than cirrhosis of the liver. The differential diagnosis of the various conditions which may produce ascites will be described at length under tuberculous peritonitis.

PROGNOSIS.—Except in the syphilitic form, the prognosis in cirrhosis of the liver is unfavorable. The progress toward a fatal ending is more rapid in children than in adults.

TREATMENT.—Curative treatment is available only in syphilitic cirrhosis; active treatment for syphilis is indicated. In other forms, treatment can only be symptomatic and palliative. Marked ascites should be relieved by paracentesis as in adults.

II. DISEASES OF THE PANCREAS

Diseases of the pancreas are practically unknown in infancy and childhood, with the exception of the general tissue-changes which may be met with in syphilis, and which have already been described. New growths of a malignant nature have been reported. The pancreas is sometimes involved in tuberculosis.

III. DISEASES OF THE PERITONEUM

The only etiological group of importance in connection with the peritoneum is that of *the infections.* Cases of various forms of new growth occurring in childhood have been reported, but they are so rare as to be pathological curiosities rather than conditions of practical importance.

Infection and inflammation of the peritoneum is seen at all ages, but in general it is less common in childhood than in adult life. For purposes of description, the following forms will be considered:

1. Acute general peritonitis.
2. Acute localized peritonitis.
3. Chronic peritonitis.
4. Tuberculous peritonitis.

ACUTE GENERAL PERITONITIS

This form may occur at any period of infancy and childhood, and has even been seen in intra-uterine life. It is comparatively common in the newborn, but after this period it is exceedingly rare during the first two years of life. It becomes increasingly more frequent from the age of two years to the age of puberty.

ETIOLOGY. THE MICROÖRGANISMS.—Acute general peritonitis may be produced by any of the pyogenic microörganisms. The organisms seen most frequently in early life are the *streptococcus* and *pneumococcus.*

MODE OF INFECTION.—In the newborn, peritonitis occurs usually as a result of direct infection through the umbilical vessels. Peritonitis may be the sole manifestation of pyogenic infection of the newborn, or may be associated with infections localized in other parts of the body. It may be a localized manifestation in the general septicemia described in Division III as infectious disease of the newborn. The microörganism usually associated with peritonitis in the newborn is the streptococcus.

In later infancy and childhood, acute general peritonitis may be primary or secondary. The primary form is rare. In cases apparently primary, the mode of infection cannot be determined. The pyogenic microörganisms may reach the peritoneum either from some remote source through the blood, or by more direct extension from some neighboring organ through the lymphatics. In some instances primary peritonitis has followed traumatism; in rare instances it has followed exposure, or a severe burn. This suggests that a local lowering of the resistance of the peritoneum may be a factor in these primary cases. It is possible that through the blood or lymphatics pyogenic microörganisms may reach the peritoneum at times, but that either because of their small number or low virulence, they are destroyed by the defensive power of the peritoneal tissue, and do not produce inflammation. If the defensive power of the peritoneum is lowered by trauma or any other means, inflammation may follow. In the light of the most recent researches, another possibility cannot be excluded in explaining the occurrence of primary peritonitis. This is the existence somewhere in the body of a focal infection, in which microörganisms of the streptococcus-pneumococcus group may acquire a selective affinity for the peritoneum. Still another possibility is direct extension from some neighboring focus of infection too minute to be found. In cases in which peritonitis follows a surgical operation upon the abdomen, the organisms may have been introduced by the operator, or may have been carried through the blood, finding the injured peritoneum a favorable soil for their development.

Most cases of acute general peritonitis are secondary, that is, are associated with some recognizable infectious process elsewhere. Here again the pyogenic organisms may reach the peritoneum by direct extension from some neighboring tissue, or by the blood from some more remote focus. The former method is much the more common. The most frequent of all the causes of acute general peritonitis is *appendicitis*. Perforation of the gastro-intestinal canal from other causes may occur, but is much less common in children than in adults. In cases due to perforation, the usual bacterial finding is the streptococcus associated with the colon bacillus.

Any abscess or suppurative focus in the tissues adjoining the peritoneum may rupture into the abdominal cavity and cause acute general peritonitis. Among the suppurating lesions most frequently extending to peritonitis in children are *empyema*, *perinephritic abscess*, and *cellulitis of the abdominal wall.* In empyema the pus may burrow through the diaphragm, but more often the infection extends through the lymphatics without rupture. Extension of the infection from the abdominal viscera to the peritoneum is much less common in children than in adults. Peritonitis is rare as a complication of typhoid fever, dysentery, abscess of the liver, ulcer of the stomach and acute intestinal obstruction. Infection of the peritoneum by extension of inflammation through the female genital tract occasionally occurs, but the process in the peritoneum is more often localized than general. Extension from the male genital organs is exceedingly rare.

Acute peritonitis may be seen as a manifestation of recognized *septicemia* or *pyemia.* It is sometimes seen as a manifestation of one of the acute infectious diseases, most frequently *pneumonia.* When peritonitis complicates pneumonia, there may be a severe pleurisy, and extension may occur through the lymphatics of the diaphragm as in empyema. In other cases the peritonitis may be part of a general pneumococcus septicemia, and in such cases meningitis or pericarditis may also be present. Peritonitis is rarely seen as a complication of scarlet fever, and of septic angina; in these cases the probable route of invasion is through the blood.

PATHOLOGICAL ANATOMY.—The lesion of acute general peritonitis is a purulent inflammation, and requires no detailed description. The peritoneum is reddened, loses its normal glistening appearance, and there is an abundant exudation of serum, fibrin and pus. In cases due to perforation, the pus usually has a foul odor.

SYMPTOMS.—The symptoms of general peritonitis in older children are much like those seen in adults, and are usually sufficiently characteristic to render the diagnosis comparatively easy. The onset is usually abrupt, with fever and vomiting. The temperature is high, 102° to 105° F. Vomiting may be present only at the onset, but more commonly continues throughout the course of the disease. The vomitus is usually green, and often contains bile.

The characteristic localizing manifestations appear very quickly after the onset. The principal symptoms are abdominal pain, abdominal tenderness and abdominal distention. Older children complain definitely of pain, and refer it to the abdomen. Younger children make no definite complaint, but manifest their pain by crying, fretfulness and restlessness. The pain is often paroxysmal. The abdomen becomes distended and tympanitic. There is usually marked rigidity of the abdominal walls, but sometimes in young children rigidity is unaccountably absent. Marked tenderness to pressure is practically a constant symptom. The pain causes the patient to assume a fixed position, usually with the thighs flexed and the knees bent. If any effort is made to disturb or move the child, it cries out with pain. The marked distension of the abdomen causes dyspnea, in which the breathing is wholly thoracic, the contraction of the accessory muscles of respiration being marked.

Children with general peritonitis appear severely ill, almost from the beginning. They have a peculiarly characteristic facies, an "abdominal" look, which is difficult to describe. The face is pinched, the eyes have a hollow, sunken look, and the mouth and features are drawn with pain. The mind is usually entirely clear. The general condition is profoundly affected. Prostration is marked, and the pulse is rapid, small in volume, and easily compressible.

The urine is diminished in quantity, and there may be retention. It may contain a small amount of albumin, and a few casts, as in any severe acute infection. Acetone is usually present, the reaction often being marked. The blood almost invariably shows a polymorphonuclear leukocytosis.

The course of the disease varies with the severity of the infection, this probably depending on the virulence of the infecting organism. In severe cases the duration is only three or four days; in milder cases a week or ten days may elapse before death occurs. As the fatal ending approaches, the patients are very apt to vomit a dark brownish material containing blood pigment. Cold extremities, cold perspiration and collapse terminate the disease.

In infants the clinical picture presented by general peritonitis is much less distinct and characteristic than in older children, and the diagnosis is consequently more difficult. Vomiting and abdominal distention occur in infants in so many other conditions, that they do not attract attention. Diarrhea often further confuses the picture. The signs of pain are not manifest. The general condition does not seem so profoundly affected in the earlier stages of the disease, although convulsions may occur. In some cases the temperature is but little elevated. Rigidity of the abdomen is occasionally absent. I have seen cases in which the only notable symptom was tenderness of the abdomen on palpation.

DIAGNOSIS.—General peritonitis in older children presents no difficulty in diagnosis. The clinical manifestations are so severe, and the localizing symptoms pointing toward the abdomen as the site of the infection are so plain, that no condition other than peritonitis is thought of as a possibility. The various functional disturbances of the gastro-intestinal tract which produce sudden attacks of abdominal pain and vomiting, do not produce marked abdominal tenderness nor such severe disturbance of the general condition. In a case of appendicitis, the question sometimes arises whether perforation and general peritonitis have already occurred. The recognition of general peritonitis in such cases depends on the sudden increase in the severity of the general symptoms, together with the appearance of general abdominal rigidity, distention and tenderness.

In infants the diagnosis is much more difficult. I have often seen general peritonitis in newborn or young infants mistaken for infectious diarrhea, or even for some functional disturbance of digestion. In such cases, diarrhea with excessive mucus or even blood in the stools, is the symptom which attracts the superficial notice of the physician. The fever and abdominal distention are attributed to intestinal infection. The principal differentiating point is the *abdominal tenderness* seen in peritonitis. This is a rare symptom in young infants, is almost never seen in infectious diarrhea, and shoudl always suggest some serious condition. Whenever an infant distinctly winces from pain on ordinary palpation of the abdomen, peritonitis should always be considered as a possibility. If the infant tends to become rapidly worse in other respects, the diagnosis is almost certain. Abdominal tenderness is seen in intussusception, but this condition is recognized by the characteristic stools and abdominal tumor.

PROGNOSIS.—The prognosis is always serious. Without surgical interference, a fatal ending is practically certain. With surgical treatment, the outlook is still serious, the prognosis depending on how early laparotomy is performed, and on the vitality of the individual child. A certain number of cases will recover if early operation is performed. The cases in which recovery has been reported without operation were probably instances of localized peritonitis mistaken for general peritonitis.

TREATMENT.—In acute general peritonitis, practically the only hope lies in laparotomy, with washing out and drainage of the peritoneal cavity. If the child's general condition is such as to afford a reasonable hope that it will withstand the shock of the operation, laparotomy should be performed at once. If the child's general condition is bad, the operation should only be delayed long enough to permit the patient to react from the ordinary measures of stimulation. The disease will not improve under medical treatment if general peritonitis is actually present. Therefore delay in performing the operation for a longer period is useless if the diagnosis is clear. The only excuse for such delay is doubt as to whether general peritonitis is present, or whether some other infection with only localized peritonitis is causing the symptoms.

In preparation for laparotomy, the most effective stimulating measure is normal saline solution given subcutaneously. From eight ounces to a pint should be given. Caffein-sodium benzoate or salicylate, and strychnin, should be given subcutaneously in doses proportioned to the age of the patient.

During the period before operation, medical treatment is wholly symptomatic. For the relief of pain, the application of cold to the abdomen by means of the ice-bag or of Leiter's coil, should be tried first. If children dislike cold applications, the hot water bag, or hot fomentations may be substituted. If severe pain continues in spite of these measures, opium should be used. Abdominal distention can be partially relieved by turpentine stupes. The best measure for the control of vomiting is gastric lavage. The diet should be milk and broths.

LOCALIZED PERITONITIS

An infection of the peritoneum, instead of producing a generalized inflammatory process, may give rise to an inflammation which involves only a part of the peritoneum.

ETIOLOGY.—The microörganisms which cause localized inflammation of the peritoneum are the same as those concerned in the production of general peritonitis. The infectious process always extends to the peritoneum from some adjoining organ or tissue. By far the most frequent source of infection in children is appendicitis. In cases of appendicitis in which perforation does not occur, there is often a certain amount of fibrinous inflammation of the peritoneum in the neighborhood of the diseased appendix. In such cases the inflammatory exudate usually remains fibrinous. In other cases of appendicitis in which perforation does occur, the same fibrinous inflammation of the surrounding peritoneum often precedes the perforation, resulting in the formation of fibrinous adhesions which so shut off the diseased organ from the general peritoneal cavity, that when perforation occurs, the result is a localized abscess.

Second in frequency to perityphilitis as a localized peritonitis, but very much less common, is subphrenic abscess. The most common source of infection is a process above the diaphragm, usually an empyema. The formation of fibrinous adhesions which later undergo fibrous organization, shuts off the area between the liver and the diaphragm from the general peritoneal cavity.

The third form of localized peritonitis is the pelvic. This is rare in childhood. It is seen almost exclusively in girls; the source of infection is the genital tract, and the organism concerned is often the gonococcus.

PATHOLOGICAL ANATOMY.—The inflammatory lesions of the peritoneum are similar to those found in inflammations of other serous membranes, such as the pleura. In the mildest form, the inflammatory exudate is chiefly fibrinous. The peritoneum is injected, and loses its glistening appearance. Considerable fibrinous exudate is thrown out, which appears as yellowish-gray patches on both the parietal and visceral layers of peritoneum. Fibrinous adhesions are formed, which may glue together various coils of intestine, or may form connections between the various viscera and the abdominal wall. In recent cases these fibrinous adhesions are soft and easily broken; in cases of longer standing they are firmer. The fibrinous exudate is usually only partially absorbed in recovery, the remainder undergoing fibrous tissue organization. The disturbances subsequently produced by these fibrous bands will be described under chronic peritonitis. In addition to the fibrinous exudate, there is usually, in the acute stage, a more or less abundant pouring out of serum, which is usually clear, but which may be turbid or bloody.

In more severe cases, the inflammation becomes purulent, and a localized abscess is formed. This is seen most often in appendicitis, but occasionally subphrenic abscess is seen in children. If the abscess is not properly drained, it may rupture into the general peritoneal cavity, producing general peritonitis, or may run a long course, eventually causing death from general sepsis.

SYMPTOMS.—The symptoms of localized peritonitis are in general like those of general peritonitis, but are less acute and severe. The onset is more gradual. The fever is apt to be less high and more irregular. Constitutional disturbance is not marked. In localized peritonitis accompanying appendicitis, the general symptoms are those of the latter.

The recognition of localized peritonitis depends upon the local manifestations in the abdomen.

APPENDIX ABSCESS.—In the form accompanying appendicitis, the formation of an appendix abscess is recognized by the finding of a resistant mass in the right side of the abdomen. This mass is usually in the right iliac region, but in young children it is often much higher up, and may occupy the right hypochondrium. The mass is tender to pressure, and on palpation may give a hard or doughy impression, or may give distinct fluctuation.

SUBPHRENIC ABSCESS.—This is a rare condition in childhood. It is most often associated with pneumonia of the right lower lobe, but it may result from an extension of infection from an abscess of the liver, or even from the appendix. The pus accumulates between the upper surface of the liver and the diaphragm, and may be very large in amount. The result is a crowding upward of the diaphragm rather than a pushing downward of the liver.

The symptoms and physical signs so closely resemble those of empyema that the differentiation is difficult or impossible upon a basis of physical examination only.

PELVIC PERITONITIS.—In this very rare form of localized peritonitis, the symptoms and signs are those of adults. There is tenderness and resistance over the lower abdomen. Rectal examination shows extreme tenderness, and often resistance or induration, in the pelvis.

DIAGNOSIS.—The diagnosis of appendix abscess is usually easy, when the process is situated in its typical position in the right iliac fossa. When the signs are higher up, the diagnosis is more difficult. When the preceding history is that of a typical acute attack of appendicitis, a tender mass in the right hypochondrium is usually a peritonitis originating in an inflammation of the appendix. If the history is that of a less acute attack, other possibilities must be considered. It should be remembered in this connection that acute cholecystitis is almost unknown in childhood. Abscess of the liver is very rare. The lesion presenting most difficulty in the differential diagnosis of appendix abscess is *perinephritic abscess.* This is not uncommon in childhood, and gives a resistance or mass in the right flank which greatly resembles that of appendix abscess. Both conditions are accompanied by fever and by tenderness to palpation. The development of perinephritic abscess is usually less acute than that of perityphlitis. Sometimes the signs of an inflammation of the kidney—a pyelitis—precede the development of the abdominal signs, and these point to perinephritic abscess. Another point well worth remembering in the differential diagnosis of these two conditions is that when the signs in the right side of the abdomen develop after a pneumonia or pleuritis, the lesion is more apt to be an abscess of the retroperitoneal tissues about the kidney, than an inflammation of the peritoneum in the right flank. This is because infection extending through the lymphatic channels of the diaphragm, if in front, causes subphrenic abscess, and if behind, causes retroperitoneal suppuration. Other conditions which may produce a resistant mass in either flank are sarcoma of the kidney, and tuberculosis of the peritoneum or mesenteric lymphnodes. In the former condition the development of the tumor is slow, the mass is not tender, and there is no fever. In the latter condition the course is also much more chronic, and tenderness is slight or absent.

The differential diagnosis between subphrenic abscess and empyema is extremely difficult. Most cases are operated upon under the impression that the condition is empyema, and the subphrenic nature of the abscess is found at operation. When there is fluid in the pleural cavity, the line of flatness both in front and behind rises higher in proportion to the amount of fluid in children than in adults. In subphrenic abscess the line of flatness to percussion and of diminished breathing does not rise so high as in most cases of empyema, and the signs resemble those found in the pleural effusions of adults. The greatest aid in differential diagnosis is to be obtained through X-ray examinations. In roentgenograms of empyema, the shadow representing the fluid occupies a large part of one side of the chest, without any very sharp upper boundary. In roentgenograms of subphrenic abscess, the outline of the diaphragm may be much higher than normal, but is usually sharply defined, and presents a more or less typical curve.

The rare cases of pelvic peritonitis seen in early life usually present little difficulty in diagnosis, which depends mainly on the results of rectal examination.

PROGNOSIS.—The prognosis in localized peritonitis is much better than that of general peritonitis. In appendix abscess, the prognosis has been considered under appendicitis. It depends a good deal upon early recognition and proper treatment of the condition. So long as the inflammatory process remains localized, the prognosis is comparatively good. The prognosis of subphrenic abscess is that of empyema. It also depends upon early recognition and proper treatment of the condition, but is not very favorable in infants. Pelvic peritonitis usually does not go on to suppuration, and the prognosis is good.

TREATMENT.—The treatment of all forms of peritoneal abscess is surgical incision and drainage. In localized peritonitis which has not progressed to suppuration, the treatment is symptomatic. The ice bag or hot water bag may be used for the relief of pain, and opiates should be given if necessary. Counterirritation is sometimes of value.

CHRONIC PERITONITIS

Under this heading are included all forms of chronic inflammation which are not of tuberculous origin.

ETIOLOGY AND PATHOLOGICAL ANATOMY.—Chronic peritonitis may occur in fetal life, and may result in extensive fibrous adhesions. These adhesions may interfere with the development of the intestine, and may produce various malformations. The cause of fetal peritonitis is not definitely known; the majority of writers have ascribed the condition to syphilis.

Chronic peritonitis may follow an infection of the newborn. I have often seen cases in which the mesentery or some coils of intestine were firmly bound, either to the region of the umbilicus, or to the liver at the point of entrance of the round ligament. These lesions represent the result of an infection through the umbilical wound.

The third cause of chronic peritonitis is a preceding acute inflammation acquired as the result of appendicitis or some similar focus of inflammation. In most cases of localized peritonitis the exudate is not all absorbed, but undergoes organization, the result being fibrous adhesions of varying extent in the peritoneal cavity.

Inflammation of the peritoneum may be chronic from the start, without any preceding acute fibrinous or purulent process. This occurs in connection with disease of any of the organs covered by peritoneum, or of organs or tissues adjoining the peritoneal cavity. In such cases a chronic inflammatory process extends from the diseased organ or tissue to the peritoneum. The result is fibrous thickening of the visceral peritoneum covering the diseased organ, or of the parietal peritoneum adjoining the diseased tissue. An example of this form of chronic peritonitis is the perihepatitis which occurs in connection with chronic adhesive pericarditis; this condition has already been described. Perihepatitis with fibrous thickening of the peritoneum may also occur in connection with chronic disease of the liver, such as cirrhosis or new growth. Chronic perisplenitis is sometimes seen. Chronic peritonitis is also seen associated with new growths of the intestines or kidneys. In all these forms the lesion may be only fibrous thickening, or fibrous adhesions may be formed. In rare cases, the form of chronic peritonitis associated with disease in the adjacent tissues may be associated with ascites, although usually ascites is only seen in chronic peritonitis of tuberculous origin.

SYMPTOMS AND DIAGNOSIS.—Most forms of chronic peritonitis have no definite symptoms, and the condition cannot be recognized clinically. It is probable that the adhesions which are formed during fetal life, or as a result of an infection of the newborn, may have some unfavorable influence upon the function of digestion. If so, the symptoms are not definite enough to be distinguished from those of common functional disturbance. Nevertheless it should be remembered that cases of apparent extreme functional disability may have an organic basis. I have frequently found at autopsies on infants dying as a result of prolonged functional gastro-intestinal disturbance, extensive peritoneal adhesions. The diagnosis of these lesions is practically impossible. Sometimes an obstinate tendency toward abdominal distention is seen, but this may be caused by so many more common conditions, that chronic peritonitis cannot appear very probable. The form of chronic peritonitis which follows a preceding acute inflammation can also rarely be recognized clinically. The presence of chronic adhesions in the peritoneal cavity is much less likely to produce subjective pain or discomfort in children than in adults. The examination of the abdomen usually reveals nothing abnormal to inspection, palpation or percussion. Sometimes a tendency toward constipation, or toward abdominal distention, is due to the existence of peritoneal adhesions, but the recognition of the condition is extremely difficult. In certain cases, careful examination with the roentgen ray will reveal certain twists or constrictions in the intestinal canal which are strongly suggestive of peritoneal adhesions. The roentgenograms are taken both after bismuth meals and bismuth enemata. A number of plates must be made before any definite conclusions can be reached.

The forms of chronic peritonitis due to extension of inflammation from an adjacent organ or tissue, are also incapable of clinical recognition. The symptoms are those of the primary disease.

The rare form of chronic non-tuberculous peritonitis with ascites presents a clinical picture almost exactly like that of tuberculous peritonitis. The first symptom noted is usually enlargement of the abdomen. Later the signs of ascites are found fully developed. Pain and tenderness are usually absent, the ascites being the only important symptom. This clinical picture means tuberculous peritonitis in the majority of cases. Non-tuberculous peritonitis can only be suspected when the von Pirquet tuberculin test is persistently negative, and when no signs of tuberculosis are found in any other part of the body. If paracentesis be performed, the fluid shows the characteristics of an exudate, not of a transudate. The fluid usually undergoes slow absorption, though at times death occurs from exhaustion or from intercurrent disease.

TREATMENT.—There is no especial treatment indicated in chronic peritonitis. In the rare cases in which the proof is plain that peritoneal adhesions are actually the cause of serious functional disturbance, operative interference with breaking up of the adhesions must be considered.

In the ascitic form, the treatment is that of tuberculous peritonitis.

TUBERCULOUS PERITONITIS

Tuberculosis of the peritoneum is one of the common clinical forms of tuberculosis encountered in early life. The pathogenesis of tuberculous peritonitis, and its relation to tuberculosis in general, has been discussed under the general description of tuberculosis.

Tuberculous peritonitis may occur at any period throughout infancy and childhood. It is one of the rarer secondary manifestations of tuberculosis in the first year of life. Most of the statistical reports bearing on the frequency of tuberculous peritonitis in infancy give the impression that the disease is commoner at this age than is really the case. This is because in these reports, based on autopsy records, every case in which there is any tuberculous involvement of the peritoneum is reckoned as tuberculous peritonitis. The peritoneum is frequently involved as part of a general miliary tuberculosis, the route of invasion being the general circulation. General miliary tuberculosis is the secondary manifestation of the disease particularly characteristic of infancy, and miliary tubercles of hematogenous origin are frequently found on the peritoneum at autopsies upon infants. But this involvement of the peritoneum as part of a general miliary tuberculosis causes no characteristic clinical symptoms, and should not be classified as tuberculous peritonitis.

True tuberculous peritonitis, with inflammatory exudate, is seen more often in the second year than in the first, but is less common than in early childhood. The disease represents a more highly developed resistance of the human organism than is seen in the acute miliary disease of infancy. It is seen most often between the ages of two and six years. After six years it becomes gradually less common throughout the remainder of childhood.

PATHOLOGICAL ANATOMY.—The tubercle bacilli gain entrance to the peritoneal cavity by direct extension, and developing there, are widely distributed over the peritoneal surfaces. Two anatomical forms are seen, the *ascitic* and the *fibrous.*

In the ascitic form the peritoneum is thickly sown with tubercles. These lesions appear as discrete and conglomerate tubercles of various sizes, without marked caseation. They are found mainly upon the visceral layers of the peritoneum, particularly on the omentum, mesentery, intestines, and under surface of the liver. In addition to these tubercles, the peritoneum shows evidences of an acute or subacute inflammation. The exudate consists of fibrin and serum. It may be mainly serous, causing an accumulation of free fluid in the abdominal cavity. In other cases there are fibrinous adhesions, causing sacculated collections of fluid in various places. The fluid is abundant, but is not so large in quantity as is seen in various other forms of ascites, such as those due to nephritis, cirrhosis of the liver, or cardiac disease. The serous exudate is usually of a clear straw-color, but it may be seropurulent. In this ascitic type, which represents a much more acute process than does the fibrous type, the mesenteric lymph nodes are usually only moderately enlarged.

In the fibrous form, there is a more chronic tuberculous inflammation of the peritoneum. The products of this inflammation have undergone more or less fibrous tissue organization, causing a thickening of the peritoneum, and extensive fibrous adhesions connecting the coils of the intestine with one another, with other organs, and with the abdominal walls. These adhesions may be the only lesions. Their presence causes a mechanical interference with peristalsis which usually leads to more or less accumulation of gas and feces in the gut. The fibrous connecting bands may be very extensive and firm. The omentum and mesentery may be greatly thickened, and in the fibrous layers matting together the structures, there may be caseous nodules or masses of various sizes, some of which may have undergone softening. In this form, the mesenteric lymphnodes are usually caseous and much enlarged. There may be some fluid exudate, either free in the peritoneal cavity, or sacculated. The exudate may be serous, seropurulent, or purulent. There may be extensive tuberculous deposits on various parts of the peritoneal surface, with caseation, or there may be tuberculous infiltration and eventual caseation of the tissues lying under the peritoneum. Such infiltration and caseation of the intestinal wall may cause perforation, or fistulous communications between various coils of intestine. Infiltration of the abdominal wall may cause a tuberculous abscess to open externally, usually in the region of the umbilicus.

SYMPTOMS. The Ascitic Form.—The initial symptoms of tuberculous peritonitis with gradual development of ascites, are usually ill defined. There is a gradual loss of appetite and flesh, with occasional abdominal pain, which, as a rule, is not severe in character. There is seldom any tenderness of the abdomen. Vomiting is not a marked symptom. Attacks of diarrhea are common and are apt to be paroxysmal. There may be constipation. The temperature is at times raised, especially in the latter part of the day, but, as a rule, is moderate, and may be so nearly normal as to suggest that the symptoms are not caused by an infection. After these general symptoms have lasted for a number of weeks, the abdomen is noticed to be distended. At first there is resonance on percussion, but later, with free fluid, there is dulness in the flanks shifting with change of position. Palpation gives a distinct fluctuation, and the "fluid wave" can be obtained. While for a time there is resonance in the region of the umbilicus when the child is lying on its back, and a change in the area of resonance with a change of position, this area of resonance gradually grows less. When the fluid is encapsulated by adhesions, the areas of dulness and resonance show a more irregular distribution, there is less shifting or no shifting with change of position, and the fluid wave across the abdomen cannot be obtained. Fluctuation, however, is present over the dull areas. As the disease progresses and the fluid increases, the whole abdomen becomes much distended and tense, the abdominal veins prominent, and fluctuation diminished. In this higher grade of ascites the umbilicus is at times found to be pushed out by the fluid.

The Fibrous Form.—This is a rather commoner form of tuberculous peritonitis than the ascitic, and is much more chronic. The onset is very insidious, and general symptoms are very vague and indefinite. Often a failure to gain weight, or a loss of weight, is the first thing to attract attention. This is usually attributed to some functional disturbance of digestion. Fever is usually entirely absent in the early stages of the disease. Vomiting is very rare. The bowels may be either loose or constipated, a common condition being constipation, with occasional attacks of mild diarrhea. The nature of the disease usually cannot be suspected until the development of localizing signs begins to point toward the abdomen.

The first localizing sign is *distention of the abdomen*. Patients in this stage of the disease are brought to the physician either because the parents have noted abdominal distention, or because of loss of weight and vague symptoms of ill-health. On physical examination, the abdominal distention is found to be due to accumulation of gas in the intestine, as the abdomen is everywhere tympanitic to percussion. There is no tenderness, and usually at this stage no masses can be felt. The distention is not very tense. It can be relieved by purgatives and enemata, but recurs at once. In some cases the abdominal distention becomes particularly marked immediately after meals. The tympanites is due to the paralysis of peristalsis caused by the fibrous adhesions between the coils of intestine.

As these adhesions contract, other symptoms appear, produced by the mechanical effect of pressure upon adjacent structures. Constriction of the intestine may produce obstinate constipation. There may be colicky pains referred to the abdomen. Pressure on the vena cava may cause edema of the lower extremities. Interference with the portal system may cause various symptoms of indigestion. and pressure on the renal circulation may cause albuminuria. General emaciation begins to be more rapid. At this stage, physical examination of the abdomen may still reveal only a condition of tympanites. The outline of the abdomen may be irregular, from the intestinal constrictions. Visible peristalsis may be present. Occasionally the fibrous bands may be palpable. The commonest abnormality to palpation found in this stage of the disease, is a rather narrow resistant mass extending transversely across the abdomen; this is usually due to rolled-up and thickened omentum.

In the majority of cases some ascites eventually develops. It is usually much less marked than in the ascitic form, and develops very slowly. The fluid is usually encapsulated, and its presence is recognized by the finding of areas of dulness to percussion with fluctuation on palpation. Fluid may be present for a time, and may then be absorbed. After absorption it may be formed again later.

The disease may remain in this stage for a long period, usually from six to twelve months, sometimes much longer. Temporary improvement, or permanent recovery may occur, without the disease advancing into its final stage.

The final stage is marked by the occurrence of extensive caseation, infiltration of the subperitoneal tissues, and softening and breaking down of the inflammatory products. This stage is characterized by well marked constitutional symptoms. There is fever, the temperature ranging from 99° F. to 102° F., or even higher. The temperature curve usually shows notable remissions and exacerbations, which may be irregular, but which often assume a typical "hectic" type, the temperature coming down each morning and going up in the evening, or the time of the exacerbations and remissions may be reversed. Emaciation is rapidly progressive, and anemia becomes marked. There is profuse sweating and marked prostration. Diarrhea is common, and at times the stools may contain blood.

On physical examination, the distention of the abdomen is often not so marked as in the earlier stages of the disease. Inspection may show irregular nodular prominences. The superficial veins are usually distended. Percussion shows areas of dulness and of tympanitic resonance irregularly distributed, but fluctuation often cannot be detected anywhere. The chief signs are found on palpation. *Nodules and irregular masses* of various sizes are felt. They may be in any part of the abdomen, but are most often found in the right iliac region or in the region of the umbilicus. The masses may form a large, hard, rounded tumor, particularly in the epigastric region, where the mass is formed by a rolled-up and greatly thickened omentum. There may be induration of the tissues of some part of the abdominal wall, with reddening of the skin, most often in the region of the umbilicus. This represents phlegmonous inflammation, and may result in the opening of a tuberculous abscess, with a fistulous communication with the peritoneal cavity.

In this stage of the disease, the resistance to the tuberculous infection is so low, that there are usually manifestations of secondary lesions in other parts of the body. Tuberculous bronchopneumonia is the most common form giving clinical symptoms in this stage. The course of the disease, after this stage of softening and breaking down has been reached, is steadily progressive. The duration is from two to six months. Death occurs from exhaustion, from tuberculous bronchopneumonia, or from tuberculous meningitis.

DIAGNOSIS.—Before considering the differential diagnosis between tuberculous peritonitis and other diseased conditions having abdominal symptoms, I must call attention to the important bearing which the presence of tuberculosis in other parts of the body has on the diagnosis of the nature of the lesions localized in the abdominal cavity. Tuberculous peritonitis is one of the secondary lesions of tuberculosis, representing an extension of the infection from the chronic lesions of the primary stage. There may be other secondary lesions. If the existence, either of primary tuberculous infection, or of other secondary lesions can be established, the probability that the abdominal symptoms are due to tuberculosis of the peritoneum is greatly strengthened.

Tuberculous peritonitis may represent the only secondary lesions, or there may be other secondary lesions. If it be the only secondary lesion, evidence can only be obtained by establishing the fact of primary tuberculous infection. The diagnosis of chronic primary tuberculosis has been considered in the article on Tuberculosis in Volume I. It depends mainly on three signs, the von Pirquet cutaneous tuberculin reaction, d'Espine's sign and interscapular dulness, and the finding of the shadows of enlarged bronchial lymphnodes in the roentgenogram of the chest. The von Pirquet reaction is usually positive in children with tuberculous peritonitis. It is occasionally negative when there is in addition some other very acute secondary manifestation, such as tuberculous bronchopneumonia or miliary tuberculosis, and also in some greatly emaciated infants. It is practically never positive unless the child is infected with tuberculosis. Consequently, a positive von Pirquet reaction is strong confirmatory evidence that the abdominal symptoms and signs are due to tuberculous peritonitis, while a negative reaction is against, but does not exclude, tuberculosis.

In many cases of tuberculous peritonitis the primary lesion is in the intestine, and in them the d'Espine's sign and roentgenogram of the chest will be negative, and will throw no light upon the diagnosis. In many other cases of tuberculous peritonitis the primary lesion is in the lungs, the bacilli reaching the abdomen through the swallowing of infected sputum. In such cases a positive d'Espine's sign, or a positive roentgenogram may be found, and if so, they strengthen the diagnosis of abdominal tuberculosis.

The commonest of the other secondary manifestations which, when present, strengthen the diagnosis of tuberculous peritonitis, is tuberculous bronchopneumonia. Other manifestations of the infection having a similar diagnostic value are tuberculous cervical adenitis, tuberculous pleurisy, tuberculous bone and joint disease, and tuberculous meningitis.

In many cases, confirmatory evidence of the presence of tuberculous infection is lacking, and the diagnosis must depend on the signs found on physical examination of the abdomen.

THE ASCITIC FORM.—This must be differentiated from other conditions characterized by ascites. The most important are cardiac disease, nephritis and cirrhosis of the liver. It should be remembered that in children the commonest cause of ascites is tuberculous peritonitis, and that the burden of proof rests on the establishing of some other condition.

Cardiac disease is recognized by the results of the physical examination of the heart. Ascites may be produced as a result of chronic passive congestion following either acute endocarditis, or chronic endocarditis with failure of compensation. In such cases, however, the ascites is usually accompanied by evidences of dropsy elsewhere, particularly in the skin of the lower extremities. There are cases of cardiac dropsy in which the edema of the lower extremities passes off before the fluid in the abdomen is absorbed. The history of a preceding edema of the skin, beginning in the feet and ankles, will usually differentiate such cases from tuberculous peritonitis, in which the fluid is confined to the abdomen. There are some cases, however, in which the pressure of tuberculous lesions in the abdomen on the vena cava produces edema of the lower extremities. There are also cases in which a clear history of preceding edema of the lower extremities cannot be obtained. In all these cases the presence or absence of signs of endocarditis are of the first importance in diagnosis.

It should be remembered, however, that the mere presence of an endocardial murmur does not necessarily mean that the ascites is due to the cardiac lesion. Many children have chronic endocarditis with endocardial murmurs, and it is quite possible that a child with such a condition might have tuberculous peritonitis in addition. If the ascites is to be attributed to cardiac disease, the physician must establish the fact, not only that the patient has endocarditis, but also that the disease of the heart is accompanied by marked cardiac insufficiency. The other signs of cardiac insufficiency are weakness of the heart sounds, enlargement and tenderness of the liver, moist râles in the lungs, and evidence in the urine of chronic passive congestion of the kidneys.

Chronic adhesive pericarditis is often accompanied by ascites without evidences of edema elsewhere. This condition can usually be recognized by the physical examination, and its diagnosis has been described under the diseases of the heart.

Nephritis is recognized by the urinary findings characteristic of that disease. Difficulty in diagnosis might arise in cases in which a tuberculous peritonitis and nephritis were both present. When ascites is due to nephritis the edema always begins in the skin, usually about the eyes.

Cirrhosis of the liver is very rare in children. When it does occur, it is very difficult to distinguish from tuberculous peritonitis of the ascitic type. In the absence of physical signs other than ascites, the diagnosis rests upon the other evidences of tuberculous infection enumerated above.

In any of the above conditions, if the ascites becomes so marked that it is considered advisable to perform paracentesis of the abdominal cavity as a therapeutic measure, the examination of the fluid obtained will at once differentiate tuberculous peritonitis, in which the fluid has the characteristics of an exudate, from other conditions, in which it has the characteristics of a transudate. Paracentesis of the abdomen for the purpose of diagnosis is never justifiable.

THE FIBROUS FORM.—When this form of tuberculous peritonitis has reached the advanced stage, the diagnosis is not difficult. The presence of a resistant band across the abdomen, of irregular nodules or masses in various parts of the abdomen, of sacculated collections of fluid, of induration or reddening about the navel—any of these signs combined with fever, rapid emaciation, and colicky pain form a sufficiently characteristic clinical picture. Nodular masses in the abdomen are sometimes due to fecal accumulations, but these can be relieved by cathartics and enemata. The epigastric tumor or thickening of the omentum is sometimes mistaken for the liver; usually, however, its upper border as well as its lower border can be determined, and this distinguishes it from the liver. It should be remembered that the absence of fever is not against the diagnosis of tuberculous peritonitis except in the final stages of the disease, because fever may be absent for a long time after local signs have become marked.

The principal difficulty in the diagnosis of the fibrous form of tuberculous peritonitis is encountered in its early stages, before the appearance of palpable masses. In this stage of the disease the only localized sign is persistent tympanites, and this may be caused by other conditions. It is seen in rickets and also in the functional gastro-intestinal disturbances of infancy. The possibility of tuberculous peritonitis must always be remembered in any patient who has developed persistent abdominal distention.

The diagnosis of tuberculous peritonitis in this stage usually depends on establishing the presence of tuberculous infection from other evidence, as described under the general diagnosis of tuberculosis.

PROGNOSIS.—Among the secondary manifestations of tuberculosis in early life, peritonitis stands about midway in seriousness. It is a less unfavorable manifestation of tuberculous infection than is acute miliary tuberculosis, or tuberculous bronchopneumonia. On the other hand, it is less favorable than tuberculous pleurisy, or tuberculous disease of the bones and joints. The prognosis varies according to the age of the patient and according to the character of the lesions in the abdominal cavity. In considering tuberculous infection in general, we have seen that the younger the child, the less highly developed is the capacity of resistance against the extension of tuberculous lesions. Consequently, the outlook in tuberculous peritonitis is grave in young children, and is particularly bad during the first three years of life. The majority of cases at this age terminate fatally. Nevertheless, it should be remembered that the outlook is never hopeless until the final stage of the fibrous form has been reached. A few children in the first three years of life recover from tuberculous peritonitis, particularly if they have the ascitic form of the disease.

The prognosis is much better in older children. The capacity of resistance against the infection is more highly developed, and many cases, if not the majority of cases in older children, make a spontaneous and complete recovery.

The outlook is very much better in the ascitic form than in the fibrous form, in spite of the fact that the former represents a more acute process than the latter. It is probable that in spite of the acuteness of the process, the rapid formation of ascites represents a higher power of local resistance on the part of the peritoneum than does the fibroid reaction of the more chronic form. In an older child with the ascitic form of the disease, and with no secondary manifestations of tuberculosis other than the process in the abdominal cavity, the outlook is very good. In an older child with the fibrous form of the disease, the process not having advanced into its final stage, the chances are perhaps about even. When the disease has reached the final stage of the fibrous form, the outlook is practically hopeless.

The prognosis is further complicated by the liability in early life to the development at any time of other more unfavorable manifestations of the tuberculous infection.

TREATMENT.—The general treatment of tuberculous peritonitis is that of tuberculosis in general, and this has been thoroughly described in the article on Tuberculosis in Volume II. The fresh air treatment should be carried out with the greatest thoroughness, and the most careful attention should be paid to the feeding and to the improvement of the general nutrition. The only modification of the general treatment of tuberculosis required by the comparative acuteness of the process, and by its localization in the abdominal cavity, is that the child should be kept in a recumbent position. The patients should be kept in bed until distinct signs of progressive improvement are manifest. They should, however, sleep outdoors and spend all their time in the open air. Under this general hygienic and dietetic treatment, a good proportion of cases of tuberculous peritonitis, particularly in older children, will arrive at complete recovery. Symptomatic treatment is rarely required. Drugs play little if any part in the treatment of tuberculous peritonitis. Pain is almost never severe enough to require opiates, and the colicky pains which are sometimes seen in the disease can be controlled by simple methods such as hot applications to the abdomen. The bowels must, of course, be kept open by appropriate measures. The tympanites is to be relieved by cathartics, enemata, and turpentine stupes; sometimes the rectal tube is required to get rid of the accumulated gas. The ascites in tuberculous peritonitis does not often become sufficiently marked to require paracentesis of the abdominal cavity. This procedure should not be employed unless the accumulation of fluid becomes sufficiently great to embarrass either the circulatory or the respiratory function.

There are three methods of treatment which at various times have been advocated as having a curative influence on the tuberculous process in the abdominal cavity. These are tuberculin injections, heliotherapy, and laparotomy.

The value of therapeutic injections of tuberculin in the treatment of chronic tuberculosis has been discussed in the article on Tuberculosis in Volume II. There is no conclusive evidence that tuberculin is of value in promoting recovery in tuberculous peritonitis. On the other hand, there is a possibility that it may be of benefit, and when properly carried out, it can do no harm. In any case of tuberculous peritonitis which is progressing unfavorably under general hygienic treatment, I should advocate the use of tuberculin, the treatment being carried out in the way described for the treatment of the chronic primary stage of the disease. (See Vol. II, page 387.)

Heliotherapy, or the exposure of the child's body to the direct rays of the sun, has been much vaunted as a treatment for tuberculous peritonitis and for tuberculous joint disease. Those who have employed this method of treatment are very enthusiastic in its favor. The treatment consists in stripping the child naked and allowing it to lie exposed in the sunlight. The exposure should at the beginning be only a few minutes in length, and the time is then gradually lengthened until its duration is three or four hours. I have had no personal experience with this treatment, but must confess that I have been favorably impressed with the reports which I have read.

Laparotomy was at one time widely advocated as a procedure having a specific influence in the cure of tuberculous peritonitis. This was at a time when the fresh air and dietetic treatment of the disease was not so thoroughly carried out as at present. The patients in whom operative treatment was employed were often sent to convalescent homes, or to other places where a more thorough fresh air treatment could be realized. I think it is for this reason that the first reports of the results of operative treatment were so favorable. More recent reports have not shown sufficient evidence of favorable results from laparotomy to warrant its use as a routine measure in tuberculous peritonitis. A comparison between cases treated by operation and cases treated by thorough hygienic and dietetic measures, has not shown any advantage in favor of the former. Nevertheless, there is a possibility that operation may be of value in certain cases. The most favorable results have been obtained with the ascitic form of the disease. I should consider operative treatment under the following circumstances: 1. In a case of the ascitic type not progressing favorably under the other available methods of treatment. 2. In cases with intestinal obstruction due to fibrous adhesions, or with severe recurrent colicky pains, showing no tendency to improve with other available methods of treatment. 3. In cases in which suppuration has occurred. It must be remembered that except in the ascitic type of the disease, a certain amount of danger attends the operation, as the fibrous adhesions often cannot be relieved except at the risk of an injury to the intestines, which may result in fecal fistulae. I have advocated operative treatment in very few cases of tuberculous peritonitis, and have seen many cases recover completely under the general treatment of tuberculosis.

DIVISION XII

DISEASES OF THE KIDNEYS, BLADDER AND GENITALS

DISEASES OF THE KIDNEYS

I. CONGENITAL MALFORMATIONS

Developmental lesions of the kidneys are not infrequent in comparison with malformations of other organs, but are rare in comparison with the diseases of the kidney due to other causes. Many of them have no practical clinical significance. *Absence of the kidneys* is very rare, is usually accompanied by other severe deformities, and is incompatible with life. *Absence of one kidney*, while less uncommon than absence of both, is still a very rare anomaly. Various defects in the development of one kidney, usually the left, are sometimes found at autopsy. Perhaps the most frequent of the rare anomalies are *fusion of the kidneys*, or horseshoe kidney. Supernumerary ureters are sometimes found.

The only developmental lesions of the kidneys which ever have any significance in practical diagnosis, are *hydronephrosis*, *congenital cystic kidney*, and *displacements of the kidney*.

HYDRONEPHROSIS

Hydronephrosis, or "water-bag kidney" is a condition of dilatation of the pelvis of the kidney, with atrophy of the renal parenchyma.

ETIOLOGY.—The condition may be either congenital or acquired. The majority of the cases encountered in early life are of congenital origin, and it is for this reason that the disease is classified among the malformations. The causes of the congenital type are malformations affecting the ureters or the urethra, such as atresia of the ureter, abnormal valvular formation, or congenital phimosis. These lesions obstruct the flow of urine from the kidney. In some cases an obstruction cannot be found at autopsy.

The acquired cases are usually not seen until later childhood. The causes are acquired conditions obstructing the flow of urine from the kidneys, such as inflammation of the ureter, concretions, or the pressure on the ureter of a tumor.

PATHOLOGICAL ANATOMY.—There is a variable dilatation of the pelvis and calices of the kidney. The mechanical effect of the pressure causes a variable amount of atrophy of the renal parenchyma. In slightly marked cases the dilatation of the pelvis is slight or moderate, and the renal parenchyma shows little atrophy, or later it may show the lesions of a chronic nephritis. In marked cases the dilatation of the renal pelvis is so enormous that a large bag filled with fluid is formed, or a number of large communicating pockets, surrounded by only a thin layer of renal cortex.

In the congenital cases, either one kidney or both may be affected. In the acquired cases the condition is usually unilateral. When the point of obstruction is in the ureter, the part of the ureter above the constriction is widely dilated. When the obstruction is in the urethra, both ureters are widely dilated throughout their entire extent.

The congenital form of hydronephrosis is often accompanied by other malformations of the genito-urinary system. There is usually also marked hypertrophy of the bladder.

SYMPTOMS.—In the congenital cases there are no symptoms other than the abdominal tumor. There are usually no symptoms in the acquired form, but there are cases on record with sudden obstruction and acute development of hydronephrosis, in which there was abdominal pain, vomiting, and symptoms of uremia.

In congenital double hydronephrosis, death occurs in early infancy. often before a diagnosis can be made. In some cases fluctuating tumors, which may reach a large size, may be felt on both sides of the abdomen in the region of the kidney. In other cases no renal tumor can be detected, but a hypertrophied bladder, appearing as a hard rounded tumor, can be made out.

In the unilateral cases, there are usually no symptoms until the appearance of a tumor. The tumor may be situated in the upper lumbar region, or may be so large as to fill the entire abdomen. The character of the tumor is recognized by the fluctuation obtained on palpation. The tumor is not tender. Urinary symptoms are usually absent until a late stage of the disease, when the urine may show the characteristics of a chronic nephritis.

DIAGNOSIS.—No diagnosis of hydronephrosis can be made until the appearance of the characteristic tumor; consequently many cases, especially the congenital bilateral forms, are not recognized during life.

A fluctuating tumor in the upper lumbar region should always suggest hydronephrosis. The other conditions giving a tumor in this region are malignant tumor of the kidney, perinephritic abscess, and congenital cystic kidney. In malignant tumor there is no fluctuation. I penrinephritic abscess there is usually fever and tenderness, and fluctuation can usually not be easily obtained. In congenital cystic kidney, fluctuation can often not be obtained, and when present it is less marked than in hydronephrosis. The diagnosis can be made positive by aspiration.

When the tumor is so large as to fill the abdomen, ascites must be excluded. In hydronephrosis the dulness does not shift with change of position. A positive diagnosis can only be made by aspiration, and depends on the fact that the fluid obtained contains urates and urea.

PROGNOSIS.—Bilateral cases are usually fatal within a year. In unilateral cases the duration of life is indefinite. There is no tendency toward spontaneous cure, and in most cases death occurs from some intercurrent infection before adult life is reached. Operation gives the only hope of relief.

TREATMENT.—In cases of unilateral hydronephrosis, in which the other kidney is normal, *nephrectomy* should be performed. In cases occurring in children over three years, the other kidney is usually normal, and this fact is shown by the secretion of a normal urine. Nephrectomy, while a grave operation, is not serious enough to be contraindicated in so grave a condition. It will bring about a complete cure. Aspiration will only give temporary relief.

CONGENITAL CYSTIC KIDNEY

This malformation, while rare, may be clinically recognizable in infants.

ETIOLOGY.—Virchow believed that the condition was one of retention cysts caused by obstruction of the urinary ducts. A more plausible theory is that the condition represents a true developmental anomaly, with the formation of cysts instead of normal renal tissue.

PATHOLOGICAL ANATOMY.—The condition is usually bilateral. The kidneys may be of normal size, with the surface studded with cysts varying from the size of a pin's head to that of a pea. The entire substance of the kidneys may consist of similar cysts held together by connective tissue. In some cases the cysts are fewer in number.

In another more rare type, the kidneys are enormously enlarged, and may fill the entire abdominal cavity and interfere with the birth of the child. In this type the individual cysts are large.

SYMPTOMS.—The type without renal enlargement shows no clinical manifestations, and is only recognized at autopsy. The second type is recognized by the presence of an abdominal tumor at birth.

PROGNOSIS.—The duration of life depends on the amount of normal renal parenchyma which is present. A few cases with small cysts have lived for a number of years. In the second type with tumor, death usually occurs in early infancy.

TREATMENT.—As the condition is usually bilateral, no treatment is available. Only in the case of unilateral tumor, with normal urinary secretion, should operation be considered.

DISPLACEMENT OF THE KIDNEYS

There are two types of displacement of the kidney seen as a congenital condition in infancy. One is *dystopia,* or downward displacement, a true developmental malformation which is extremely rare. The left kidney is the one usually involved. The practical significance of the condition lies in the fact that the displaced kidney is palpable as an immovable tumor low down in the abdomen. There are rare instances on record in which such a tumor has been confounded in differential diagnosis with pelvic new growths, fecal concretions, enlarged glands, appendicitis, and intussusception. The results of careful palpation are important in establishing a diagnosis in such cases.

The other type of displacement is *movable kidney.* This is more common than dystopia, but nevertheless the congenital form, which is the only one seen in early life, is very rare. The cause is probably a developmental anomaly involving the mesentery and renal blood vessels, which results in the kidney having too long a pedicle. The condition may be unilateral or bilateral; it is most often unilateral, and usually involves the right kidney.

SYMPTOMS.—The condition may exist without any symptoms, and be discovered accidentally at any time throughout childhood in the course of an abdominal examination. There is sometimes a feeling of heaviness and pressure in the abdomen. Attacks of pain radiating into the extremities, appearing most often after a mechanical jar, are sometimes seen, but the severe paroxysms with collapse and fainting, due to torsion, are practically unknown in early life.

DIAGNOSIS.—The diagnosis is made by palpation. In spite of the relatively large size of the kidney in early life, the organ is not normally palpable. In floating kidney palpation reveals a movable, smooth, oval tumor which can easily be pushed into its normal position.

TREATMENT.—The hygienic surroundings of the patient should be regulated, with careful attention to diet, bowels, fresh air, exercise, and so forth. The kidney after being replaced must be retained in normal position by a well-fitted abdominal belt. Operation is rarely required in early life.

II. NEW GROWTHS

The tumors of the kidney encountered in early life are usually malignant. Occasionally benign tumors not large enough to be clinically recognizable, are found at autopsy. In very rare instances a clinically recognizable tumor of the kidney may prove to be of a benign form.

MALIGNANT TUMORS OF THE KIDNEY

ETIOLOGY.—Malignant tumors of the kidney are more common in childhood than in adult life. The majority of cases are seen in early childhood. The new growth may be congenital.

PATHOLOGICAL ANATOMY.—In the great majority of cases, the new growth is some variety of *sarcoma.* Carcinoma is extremely rare. Birch-Hirschfeld groups the malignant tumors of the kidneys under the term *embryonal adenosarcomata.* The hypernephroma, originating in adrenal cells embedded in the kidney, must be included among the varieties found.

The new growth may start in the cortex or pelvis of the kidney, or in the adrenal. It may invade the whole kidney, leaving no remaining trace of renal tissue, or it may grow from one side of the organ. A very large irregular tumor is usually formed. The tumors are often soft, but are rarely cystic. There may be secondary metastases in the liver, lungs, retroperitoneal glands, intestines, pancreas, or in the other kidney.

The growth of the tumor usually causes a localized chronic fibrous peritonitis, and the tumor may become adherent to all the neighboring abdominal organs. The weight of these tumors is usually from six to fifteen pounds, but larger growths have been reported.

SYMPTOMS.—The only characteristic clinical manifestation is the tumor, and enlargement of the abdomen is usually the first sign suggesting disease which is noted by the child's parents. Occasionally hematuria is the first symptom noted. The parents often discover the solid tumor themselves before bringing the child to the physician.

In the early stages the tumor is found in the loin. Its growth is rapid, and it grows forward toward the median line. On palpation the tumor is most often solid, but occasionally is soft, although distinct fluctuation is never detected. The mass is usually irregular, rounded, and does not have a sharply defined border. The surface may be smooth or irregular. There is no tenderness. Before the tumor has attained a very large size it is usually possible to deter-

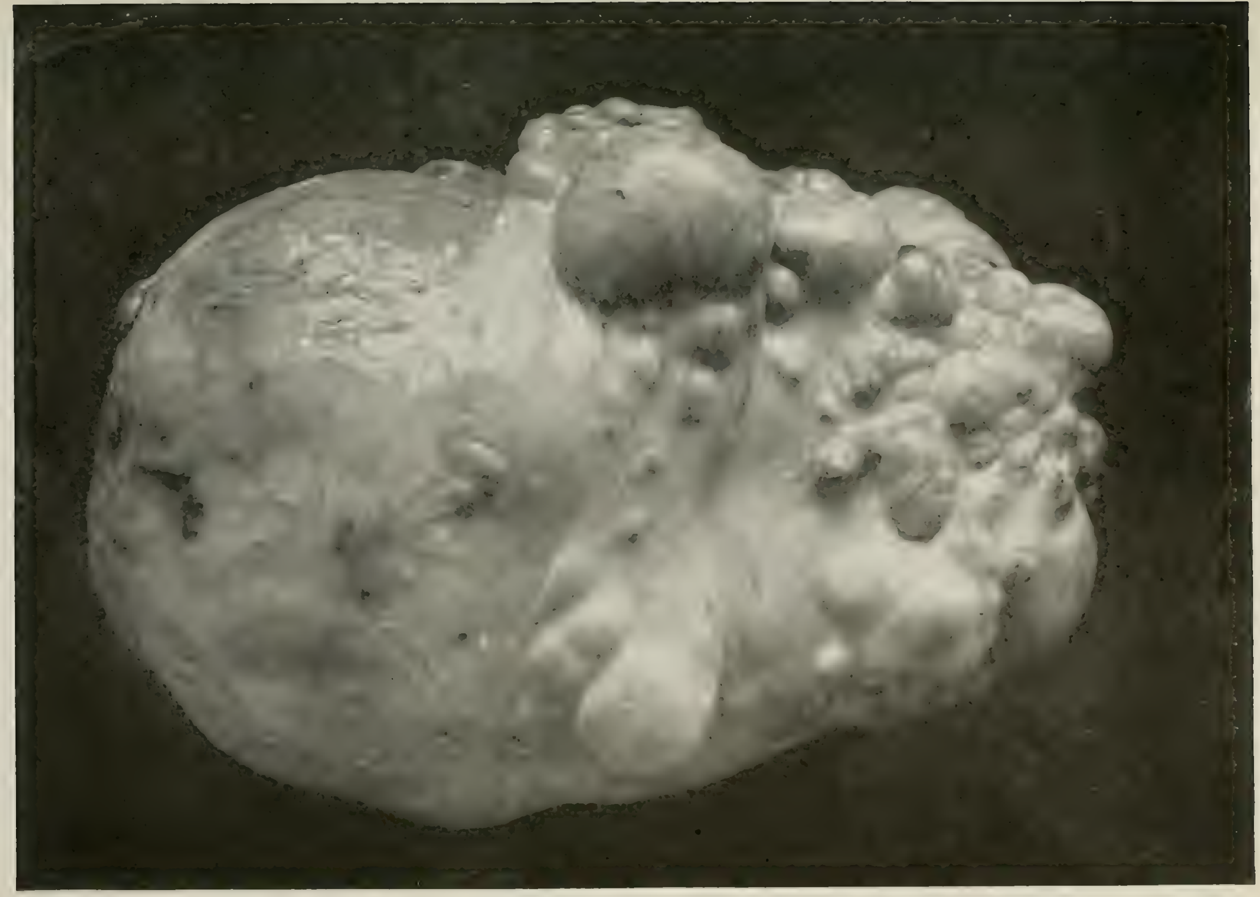


FIG. 238.—Sarcoma of the Kidney. Weight 6 pounds, removed from an infant eight months old.

mine by careful palpation that, although the mass may be movable, its point of attachment is near the spine in the lumbar region.

As the tumor grows, it occupies more and more of the abdominal cavity, and may fill almost the entire abdomen, displacing the liver, spleen, intestines and lungs. In this stage a much enlarged abdomen is found, in which palpation immediately discloses the enormous solid mass.

Hematuria is a common symptom, and may be present in any stage of the disease. The urine may be macroscopically bloody, or the amount of blood may be so small that it can only be detected with the microscope. Pain is a rare symptom. In the later stages of the disease, cachexia develops rapidly, while the tumor continues to grow.

Various symptoms may be produced by pressure of the tumor upon adjoining structures. The most common are symptoms of indigestion, cough, and edema of the lower extremities.

The disease runs a fairly rapid course, and progresses steadily toward a fatal ending from cachexia and exhaustion. In cases not operated upon, the duration of the disease from the time the tumor is first discovered varies from two months to a year.

DIAGNOSIS.—Malignant new growth is the only common cause of a solid tumor in the abdominal cavity. The important points in favor of a diagnosis of sarcoma of the kidney are the age of the patient (most cases are under five years), the solid character of the tumor, its point of attachment in the lumbar region, its steady and comparatively rapid growth, and the occurrence of hematuria.

The only difficulty in diagnosis arises from the fact that the physician cannot always be certain that the mass felt on palpation represents a tumor connected with the kidney. The conditions which most commonly have to be considered in differential diagnosis, are enlargement of the spleen, tumor of the liver, a mass of tuberculous mesenteric lymphnodes, hydronephrosis, perinephritic abscess, and dermoid cyst of the ovary.

Enlarged Spleen.—This may cause a large mass to be felt on the left side of the abdomen. The tumor caused by an enlarged spleen is more superficial, and has a smooth surface, and a defined edge, whereas the tumor of renal sarcoma appears to extend deep into the abdominal cavity, often has an irregular or nodular surface, and has an irregular outline without well-defined borders. A point of great importance is that if any superficial tympanitic resonance on percussion can be obtained over the tumor, it is a renal growth, not an enlarged spleen. This is because an enlarged spleen pushes aside the intestines and lies directly under the abdominal wall, whereas a renal tumor cannot push aside the descending colon, which is attached to the surface of the growth unless there be a liberal mesocolon.

Tumors of the Liver.—These are much more uncommon in early life than are tumors of the kidney, but when present and deep-seated, they often give rise to great difficulty in diagnosis. The possibility of diagnosis depends upon whether the physician, by careful palpation, can make out a definite connection of the tumor with the substance of the liver. In sarcoma of the kidney there is often a palpable depression between the mass and the edge of the liver, which is of great help in diagnosis. If hematuria be present, the diagnosis of renal sarcoma is confirmed. In cases of doubt in children, it is better to consider that tumors are probably of renal origin, unless they can definitely be connected with the liver.

Tabes Mesenterica.—A mass of tuberculous lymphnodes, or a thickened omentum in tuberculous peritonitis, may present a palpable tumor in the abdominal cavity. The tumor caused by a thickened omentum is rarely in a situation such that it could be mistaken for a renal growth. A mass of tuberculous lymphnodes is usually more movable than renal sarcoma, but the diagnosis may be very difficult, depending upon whether there be hematuria on the one hand, or other evidences of tuberculous infection on the other. Of course during the period subsequent to the first observation, the steady growth of the mass, and the large size attained, clearly reveals the nature of the disease.

Hydronephrosis.—The differentiation of this condition from renal sarcoma depends on the distinct fluctuation which is obtained on palpation, and on the absence of hematuria.

Perinephritic Abscess.—In this condition the outlines of the mass are even less defined than in renal sarcoma. The mass is usually tender to palpation, and fever is usually present.

Dermoid Cyst of the Ovary.—This is very rare in early life. The diagnosis depends on the ability to recognize on palpation that the tumor originates in the pelvis.

PROGNOSIS.—The prognosis is hopeless without surgical interference.

TREATMENT.—Operation, with removal of the growth, is indicated in every case in which the diagnosis of malignant tumor of the kidney is made. The operation is a serious one, and has a fairly high immediate mortality. This is because in many cases the difficulty and shock of the operation are much increased by the adhesions which have formed between the tumor and neighboring organs. Of the cases which survive the operation, in a certain number recurrences occur in the opposite kidney, or death occurs from metastases into other organs. Nevertheless the number of cases in which permanent recovery has followed the operation has been sufficiently great to warrant this treatment as a routine in so fatal a disease.

III. FUNCTIONAL DISTURBANCES

The multiple factors acting from outside the body, such as unfavorable conditions of hygiene and environment, have less influence upon the function of the kidneys than upon that of other organs of the body, such as, for example, those of the digestive or nervous systems. For this reason there are few diseases which are primarily disturbances of renal function. The function of the kidneys is disturbed mainly by the systemic effects of other well-recognized diseased conditions, and the clinical manifestations of functional renal disturbance are mainly secondary, and appear as symptoms of the primary disease. In diabetes insipidus it is an open question whether or not this disease represents a primary disturbance of renal function, or whether it represents a disturbance secondary to a general constitutional disturbance or to a disturbance of the function of the nervous system. Orthostatic or cyclic albuminuria probably represents the most typical primary disturbance of the function of the kidneys. The only other pathological process which represents a primary disturbance of the renal function is the formation of renal calculi. The uric acid infarction observed in newborn babies is a normal physiological process, which only occasionally gives rise to symptoms of functional disturbance. It is thus evident that the primary functional disturbances of the kidney are comparatively few. The chief function of the kidney is the secretion of the urine. The most common functional disturbances of the kidney are anomalies of the urinary secretion, and these are usually secondary to recognized diseases.

ORTHOSTATIC ALBUMINURIA

(Cyclic Albuminuria) (Paroxysmal Albuminuria)

(Functional Albuminuria) (Albuminuria of Adolescence)

The presence of albumin in the urine is usually associated with an organic lesion of the kidney, such as is seen in the various forms of nephritis of toxic or infectious origin. Even if we leave aside those cases in which the presence in the urine of some albumin-containing fluid, such as blood or pus, gives a positive clinical test for albumin, albuminuria of renal origin is not always a sign of an organic lesion of the kidneys. It may represent a purely functional disturbance.

In considering functional albuminuria, it is very difficult to draw a sharp line between the normal and the pathological. It always represents a functional disturbance, but the causes in many cases are so slight, that the presence of albumin in the urine cannot really be considered a manifestation of disease. At the other extreme are cases in which albuminuria without organic lesions of the kidney is associated with multiple other signs of disease. Between these two extremes are every variation in severity.

Transient albuminuria is seen at times after excessive physical exertion, after relative over-eating, after cold baths, and after excessive emotional excitement. It has been produced by the palpation of normal kidneys. In a certain number of normal children, during the period of approaching puberty, there is a form of albuminuria which comes and goes with a certain periodic regularity. The repeated finding of albumin in the urine of such cases is strongly suggestive of nephritis, but the entire absence of other signs of disease, and the uniformly favorable prognosis, makes the recognition of the functional nature of the albuminuria a matter of great importance.

Various names have been applied to this recurrent transient form of albuminuria. Pavy, its discoverer, called it *cyclic albuminuria*, on account of the tendency of the albumin to appear only in the urine passed at a particular time of day. If a normal child of an age approaching that of puberty be made to kneel in an upright position, a transient albuminuria will often appear. As in the recurrent cases the albumin is usually found in the urine passed toward the end of the day, its appearance would seem to be connected with the upright position. Stirling first called the disturbance "*postural albuminuria*." The term most commonly employed at the present day, "*orthostatic albuminuria*," has the same origin. This name lays stress on the upright position as an etiological factor, but this is probably not a factor in all cases. It is German writers in particular who have laid stress on the postural element in etiology. French writers have insisted on a sharp differentiation of the orthostatic form from other forms of transient albuminuria, recognizing not only a "cyclic" form not due to posture, but also a "hepatogenous," "digestive," and "pretuberculous" form.

ETIOLOGY. OCCURRENCE.—Orthostatic albuminuria is seen most often in later childhood, rarely occurring except between the ages of seven and fourteen years. It is most common between eleven and fourteen. The number of girls predominates over that of boys. The majority of cases are probably overlooked, because routine examinations of the urine of children apparently normal are rarely made. German figures based on the investigation of a series of school children give the frequency of occurrence as varying from 5 per cent to 30 per cent.

The element of heredity has been observed in numerous instances.

The occurrence of more than one case in a family is not rare. The disease is commoner in the children of the proletariat than in those of the well-to-do. Tuberculosis and neuropathy are the principal positive findings in the family history.

PATHOGENESIS.—While some light has been thrown on the etiological factors which produce this functional disturbance, many questions remain unanswered. The weight of evidence is decidedly against the view that the disturbance represents a very slight mild grade of nephritis.

The view of the pathogenesis of the disease which is most generally held, is that in typical cases the appearance of albumin in the urine is caused by a *change from the recumbent to the erect posture.* This was suggested by the usual absence of albumin in the morning urine, and is supported by certain convincing experimental evidence. It has been found that recumbency during the day prevents the appearance of the albuminuria, but it reappears when the child gets up. It has further been found that if the patient gets up, but the body is supported by a hanging attachment to the head, the albumin does not appear. This suggests that it is not merely the erect position of the body, but the *holding* of the body erect, which is of etiological importance. Jehle found in most of his patients a certain amount of spinal lordosis. The normal holding of the body erect brings about a certain amount of lordosis in the lumbar region. Jehle's theory was that this lordosis produces a functional disturbance of the kidneys by mechanical action on the renal circulation.

There can be no doubt that lordosis, or the position of the spine when the body is held erect, is an important factor in the etiology of typical cases of cyclic albuminuria. Lordosis produced artificially has caused the appearance of albumin in the urine of healthy children. On the other hand, there are cases of marked lordosis which do not show albuminuria. Lordosis is certainly not the only factor in the etiology of the disease.

The occurrence of cyclic albuminuria in some children, and its absence in others can only be explained by another factor, one involving the general constitution of the child. This constitutional factor is apparently present only at a certain age. Little is known as to the nature of such a constitutional factor. Among the causes which have been advanced as important in the constitutional tendency toward functional albuminuria are the following: (1) A general asthenic condition of the child, possibly produced by the rapid growth seen at this period of life, and acting on the kidneys through the vaso-motor system. (2) A relative functional cardio-vascular insufficiency, with a relatively small heart and arteries, which condition is common at this age. (3) A hereditary tendency to functional incapacity of kidneys. (4) Tuberculosis, or other chronic infections.

I would summarize as follows the most probable theory of the etiology of orthostatic albuminuria. The condition represents a functional incapacity of the kidney produced by a constitutional condition. The constitutional condition probably has multiple causes. Acute infection may be a contributing cause. The immediately exciting cause is most often the mechanical effect of the slight lordosis produced by the erect posture. In some cases with albuminuria, which are not so typical clinically, lordosis is not the immediate cause, which is unknown.

SYMPTOMS.—There are no symptoms peculiar to or characteristic of the disease, except the albuminuria. The symptoms which are present, and which are the cause of the patient being brought to the physician, are either those of some other definitely recognizable disease, or else are those of the constitutional peculiarity or abnormality which acts as the underlying predisposing cause of the functional disturbance of the kidneys. In the last years of childhood, growth is very rapid, and children are apt to outgrow their strength. The combination of a hereditary predisposition and this rapid growth, plus possible unfavorable features in the hygiene and environment of the child, gives rise to a certain amount of functional disturbance in all parts of the body. It is the symptoms of these functional disturbances which are complained of.

Among the common symptoms of general functional disturbance in late childhood are headaches, a feeling of weakness, loss of desire to play or study, attacks of dizziness, occasionally even attacks of syncope, loss of appetite, occasionally morning nausea, attacks of palpitation of the heart, "stitches" in the side, pains in the back or limbs, nosebleeds, nervous irritability and many other complaints of subjective sensations. When the physician is confronted with a history of any of these symptoms, it is his duty to make a thorough examination for the purpose of determining whether any organic disease is at the bottom of the symptoms, and if not, what errors in hygiene are responsible for the functional disturbance.

When no evidence of definite organic disease is found, physical examination often reveals various peculiarities. The patient may simply be a child too large for its age, but otherwise normal. The child may be thin, with poorly developed muscles, and may appear pale without the blood showing the characteristics of anemia. The various manifestations enumerated under functional disturbances of the heart are common, and there is often evidence of poor peripheral circulation. Systolic murmurs without cardiac enlargement or other evidence of an organic lesion are common. The blood pressure is usually normal, but the pulse is often of small volume.

In every case of this kind a thorough investigation of the cause of the symptoms should include an examination of the urine. If only a single specimen be examined, the albuminuria is quite as likely to be missed as to be found. In all cases a specimen of the total quantity passed in the twenty-four hours should be examined This will show a positive test for albumin, if functional albuminuria be present. I have been called to see a number of cases for nephritis because albumin was found in a particular single specimen, or in the mixed twenty-four hour quantity. Other cases have been sent to me because the physician, after repeated examinations of single specimens, found albumin sometimes present, sometimes absent.

The urine shows no abnormality of gross appearance, daily quantity, or specific gravity. The sediment often shows a deposit of phosphates or urates. In girls there is often mucus, squamous epithelium, and leukocytes from the genital tract. Renal casts are usually absent, but sometimes are found in the specimens containing albumin, so that their presence does not necessarily mean nephritis.

In order to arrive at the correct diagnosis, the physician must order that each specimen of urine passed be saved separately, and the time of passage recorded on a label. The examination of these separate specimens will show that each day albumin is present in some specimens, absent in others. The specimen most likely to contain albumin, and containing the largest quantity, is the second passed after the patient gets up in the morning. The first morning specimen passed as soon as the patient gets up is usually free from albumin, especially if the patient has urinated without getting out of bed during the night. Occasionally the secretion of albumin may continue a short time after the patient goes to bed, and if no urine be passed during the night, a very slight trace may appear in the first morning specimen. The presence or absence of albumin during the rest of the day depends on the manner of life of the patient. If the child is continuously up and mainly on its feet, the quantity of albumni diminishes, and the specimens of urine passed toward night are apt to contain none. If the patient is particularly active at any period of the day, the urine secreted during this period is likely to contain more albumin. If the patient is recumbent for part of the day, the urine secreted during the period, or at least during the latter part of this period, will contain no albumin. When the patient gets up after recumbency, the urine secreted during the period right after getting up will again contain a maximum quantity of albumin. Careful separation and examination of the several specimens will reveal that in typical cases it is the change from a recumbent to an upright position which is the chief cause of the appearance of albuminuria. During prolonged recumbency, albuminuria will remain absent. The following table shows the urinary findings in a typical case of orthostatic albuminuria:

TABLE 68

Two Days' Urinary Findings in Orthostatic Albuminuria

DATE	HOUR	ALBUMIN
May 5	8.00 A. M.	0
	12.00 M.	Trace
	1.00 P. M.	0
	6.00 P. M.	Slight trace
	10.00 P. M.	Slight trace
May 6	7.30 A. M.	0
	10.00 A. M.	Large trace
	1.00 P. M.	Slight trace
	6.30 P. M.	0
	10.30 P. M.	0

The quantity of albumin is small, varying from a slight trace to .1 per cent. The maximum is most often described by the term "large trace." While the ordinary serum-albumin is always present, a trace of nucleo-albumin is usually also present. This is shown by a slight clouding when acetic acid is added. Serum-albumin is recognized by increased cloudiness on boiling, or by giving an increased cloudiness with potassium ferrocyanid.

DIAGNOSIS.—The diagnosis depends upon the peculiar "cyclic" character of the albuminuria described above.

PROGNOSIS.—The recognition of orthostatic albuminuria is important from the point of view of prognosis rather than of treatment. The prognosis of orthostatic albuminuria is good. The chief function of the physician is to relieve the minds of the child's parents, who often have been much frightened by the suggestion that the patient has Bright's disease. Of course the certainty with which a favorable prognosis can be given depends upon the certainty of the diagnosis. While most cases having orthostatic albuminuria have been thought to have nephritis, a few cases have been diagnosed as orthostatic albuminuria which subsequently developed a true nephritis. When the albuminuria shows the typical peculiarities described above, the favorable prognosis need not be qualified. When the albuminuria is "cyclic," but does not show a relation to the change from a recumbent to an upright position, the probabilities are still strongly in favor of its functional character, but there is a slight possibility of nephritis, especially if casts be present. When albumin is constantly present in the urine without other evidence of nephritis, the diagnosis and prognosis are doubtful.

TREATMENT.—The treatment is principally that of the underlying constitutional condition. Every feature in the surroundings and daily life of the patient should be scrutinized, with a view to discovering and correcting all possible errors. The diet should be simple, and the daily life of the child should be that which best conduces to rugged physical development.

Although prolonged rest in bed will cause a complete disappearance of albumin from the urine during the period of recumbency, such a method of treatment is most decidedly contraindicated. The essential point in treatment is not to prevent albuminuria, but to improve the underlying constitutional condition. Preventing albuminuria does no good, and as soon as the patient gets up, albumin will reappear, perhaps in larger amount than before. Similarly, although exercise may increase the albuminuria, it is not contraindicated. The normal activities of a child of this age should include a large amount of exercise, which is necessary for proper physical development. It is not a good plan to restrict exercise to an amount below the normal. Gymnastic exercises under proper supervision are valuable, but should not be allowed to take the place of exercise in the open air. I allow patients with orthostatic albuminuria to engage in all the games and sports proper for normal children of the same age and physical development. Of course some restriction in the violence of the exercise allowed must be made in cases with relative functional insufficiency of the heart. The only restrictions peculiar to orthostatic albuminuria which I recommend are the forbidding of bicycle riding and horseback riding, both of which require that the back be held in a more or less fixed position.

If the physician remembers that orthostatic albuminuria requires no specific treatment, that the great object of treatment is to improve the general condition and favor rugged physical development, and modifies details to suit individual constitutional peculiarities, the functional disturbance will disappear, usually within a year.

LITHURIA. URIC ACID INFARCTION

The term lithuria is applied to a condition in which there is an excess of uric acid and its salts in the urine. One peculiarity of the urine in early infancy is that it contains a relatively small quantity of urea, and a correspondingly large quantity of uric acid. In some forms of animal life, uric acid is one of the principal end-products of protein metabolism, and the same peculiarity apparently exists in the metabolism of the human fetus.

This accounts for a condition nearly always found at autopsy in the kidneys of newborn infants, and of infants dying in the first ten days of life, and which is often found throughout the earlier weeks of infancy. It is called the *uric acid infarction*, and consists in a deposit in the straight tubules of the kidneys of uric acid or urates. The uric acid infarction is visible to the naked eye, appearing as fine yellow, orange, or brownish-yellow radiating striae. There may also be granular deposits of urates in the pelvis of the kidney, sometimes with evidence of catarrhal inflammation, and rarely with blood. The cause of the condition is a secretion of the uric acid bodies in excess of the amount of water necessary to keep them in solution.

The uric acid infarction though a normal condition, may be so marked as to give rise to *symptoms*. The most common symptom is a very scanty secretion of urine in the first few days of life. Occasionally the condition may cause anuria for the first one or two days of life. In severe cases there may be evidences of pain, especially on urination, the pain being caused by the passage of undissolved uric acid bodies which may even be small calculi. Priapism is seen at times. The urine passed is highly acid, causes a reddish staining of the napkin, and may contain albumin, casts, epithelial cells, leucocytes, blood corpuscles and urates.

When the uric acid infarction of the newborn is sufficiently marked to become a pathological condition, the *treatment* is to give water freely, and an alkaline diuretic. I usually give acetate of potash, in doses of one grain every two hours. The treatment is continued until the urinary secretion is fully established.

With the progress of normal development, the tendency toward a relatively excessive formation of uric acid rapidly diminishes. Nevertheless there is throughout infancy a tendency toward excessive uric acid secretion at times. This anomaly of urinary secretion is closely connected with disturbances of digestion, nutrition and metabolism. It manifests itself by no symptoms other than the appearance of a reddish stain upon the napkins of the infant. The stain, while not closely resembling blood, is often mistaken for blood by the infant's mother or nurse, and it is sometimes necessary for the physician to reassure her as to the significance of the appearance.

There are some children who show throughout infancy and childhood a tendency toward attacks of highly acid urine in which the urates are in marked excess. This appears to be due to a constitutional peculiarity involving metabolism, in which heredity may be an important factor. Various external conditions in hygiene and environment may act as contributing or precipitating causes for the attacks, but no definite connection with the diet has been established. The condition is most often seen in children who are thin, nervous, mentally precocious, and irritable.

The *symptoms* of these attacks are nervous irritability, retention of urine, and pain on urination. The patients are very fretful; the urine is held as long as possible, and finally passed with a fit of crying or complaint of severe pain. The urine may be so irritating that the external genitals are reddened and swollen; a mild catarrhal vulvovaginitis may appear in female infants. The *diagnosis* is made by examination of the urine, which is very markedly acid, and often shows a heavy deposit of crystalline or amorphous urates. The *treatment* consists in giving plenty of water, and acetate of potash in doses appropriate to the age of the child. If the acidity of the urine is not sufficiently reduced, the doses must be increased. General regulation of the diet and hygienic surroundings of the patient is an important part of the treatment.

In some cases a highly acid urine is the cause of enuresis. This will be considered under that disease.

RENAL CALCULI

Small renal calculi are commonly found at autopsies upon infants. They rarely produce symptoms. If any clinical manifestations be present, they are those described under lithuria.

Large renal calculi are extremely rare in infancy. It is generally supposed that they are almost never found throughout childhood. This is not true. While much less common than in adults, stone in the kidney with its characteristic symptoms is occasionally seen in early life.

ETIOLOGY.—There appears to be an endemic element in the etiology, as the occurrence of calculi is commoner is some localities than in others. This may depend upon the amount of calcium in the drinking water, which has been considered an important etiological factor. Another important factor is undoubtedly that of a gouty heredity. There are probably other obscure factors involving the general metabolism, and dependent upon conditions of hygiene and environment.

PATHOLOGICAL ANATOMY.—The calculi may be composed of uric acid, phosphates, or calcium oxalate. Uric acid stones are the usual form found in children. Cystin stones are exceedingly rare. The stones cause a catarrhal inflammation of the pelvis of the kidney.

SYMPTOMS.—The principal symptom of stone in the kidney is pain, which may be referred to the affected kidney, but which is quite as likely to be referred to the bladder, to the perineum, and even to the opposite kidney. There may be some fever from the associated pyelitis. The urine may be passed frequently in small amounts.

On physical examination there may or may not be tenderness on deep palpation in the lumbar region. There may be retraction of the testicle. The urine may show pus cells and epithelial cells from the pelvis of the kidney, may contain traces of blood at times, and may contain uric acid or other crystalline deposit.

When a stone of considerable size passes from the kidney to the bladder, renal colic is produced. The attack is the same as in adults, a sudden paroxysm of severe pain in the loins, radiating to the testicle and down the thigh. There may be nausea, vomiting, or even collapse. The urine is passed frequently, and usually contains blood. When the stone reaches the bladder, the symptoms subside rapidly. Impaction of the stone in the ureter may cause hydronephrosis, which soon becomes pyonephrosis.

DIAGNOSIS.—Stone in the kidney should always be suspected from the symptoms mentioned above. When typical renal colic is present, or when the attack is accompanied or followed by hematuria, the diagnosis is strengthened. Whenever the presence of renal calculi is suspected, the total twenty-four hour urine should be saved and examined, and this must be repeated many times before stone in the kidney can be excluded. The finding in the urine of definite concretions, no matter how small, makes the diagnosis positive. Another procedure of the greatest value in the diagnosis of renal calculi is examination with the Roentgen ray, which should be made in every suspected case. In children, even small calculi will often appear clearly in the plates, although a negative X-ray does not exclude calculus.

PROGNOSIS.—The prognosis when the calculi are small, without evidences of pyelitis, and with mild symptoms, is good, with medical treatment. Severe cases, with large calculi, or evidences of pyelitis or impaction, are more serious, and without surgical treatment will result in serious nephritis.

TREATMENT.—Medical treatment consists in giving a large quantity of fluid, the alkaline mineral waters, and a quantity of potassium acetate or citrate sufficient to make the urine alkaline. Only mild cases can be relieved in this way. Severe cases require surgical operation.

Surgical interference is indicated under the following conditions: (1) When the symptoms are marked; (2) when signs of pyelitis do not improve, or increase under medical treatment; (3) when a second attack of renal colic occurs under medical treatment; (4) when there are evidences of impaction in the ureter; (5) when the X-ray shows a large calculus in the kidney.

SECONDARY AND SYMPTOMATIC FUNCTIONAL DISTURBANCES

CHRONIC PASSIVE CONGESTION.—This condition occurs in organic cardiac disease, in chronic pulmonary disease, and when there is mechanical obstruction to the venous circulation, as from pressure of abdominal tumors on the vena cava or renal veins.

PATHOLOGICAL ANATOMY.—The kidney is enlarged, firm, and of a deep-red color. The substance is tough from increase of interstitial tissue. The vessels are congested and their walls become thickened in long-continued cases. The epithelium of the tubules shows granular or fatty degeneration.

SYMPTOMS.—The symptoms which occur in the course of passive congestion are only slightly referable to the kidney, and depend for the most part upon the disease which causes the condition.

URINE.—The urine in this condition is high-colored, strongly acid, and often considerably diminished in amount. It usually has a high specific gravity, and often a heavy sediment of amorphous urates. The total solids are diminished absolutely, but owing to the concentration of the urine are relatively increased. There is a slight trace of albumin, usually under one-tenth of one per cent. Microscopic examination shows a few hyaline casts with renal cells adherent and an occasional blood corpuscle. There are, however, very few of these elements in the sediment. If the urine is passed in larger quantities, it is not so highly colored and contains a smaller amount of albumin.

DIAGNOSIS.—The diagnosis of passive congestion usually cannot be made from the urine alone. The main reliance must be placed upon the clinical history and physical examination.

PROGNOSIS.—The prognosis in cases of passive congestion of the kidney depends upon the cause of the condition.

TREATMENT.—The treatment is to be directed to the cause or causes of the congestion.

ANURIA.—Anuria is an arrest of the secretion of urine. It must be distinguished from retention of urine in the bladder, from the scanty urine passed when liquids are withheld or refused in sickness, and from diminished secretion dependent upon a profuse watery diarrhea. Total suppression of urine may occur in the intense congestion of acute nephritis, in poisoning by lead, phosphorus, or turpentine, or after severe injuries or operations.

Anuria in the newborn may be due to malformation of the urinary tract or to uric acid infarction. Other cases in infants are evidently of nervous origin, and may last for twenty-four to thirty-six hours without other symptoms. Hysterical anuria may occur in older children. Charcot reports a case lasting for eleven days.

TREATMENT.—The obstructive cases demand operation. In nonobstructive cases the treatment is usually simple and satisfactory. Sweet spirits of nitre or citrate of potash given with plenty of water will usually relieve the condition. Hot applications over the kidney may be used, or large hot irrigations of the colon with salt solution, or in extreme cases the subcutaneous injection of salt solution.

The treatment of the form of anuria which is symptomatic of toxic nephritis, will be described under that disease.

POLYURIA.—The secretion of an increased quantity of urine is seen when an increased quantity of fluid is being taken, when a transudate or exudate is being absorbed, as a symptom in some forms of toxic nephritis, as a symptom of diabetes mellitus and diabetes insipidus, and as a symptom of certain functional disturbances of the nervous system.

HEMATURIA.—This term is applied to the presence of red blood corpuscles in the urine. The commonest cause of hematuria is toxic nephritis, especially the forms which are secondary to such infectious diseases as scarlet fever, tonsilitis, typhoid fever, influenza, malaria, general sepsis, and so forth. It is also seen in the toxic nephritis caused by various forms of drug poisoning, and indeed, is a common symptom of all forms of toxic nephritis.

The commonest local cause of hematuria, especially in infancy, is malignant new growth of the kidney. It is rarely due to a new growth in the bladder. There are rare instances of hematuria due to traumatic injury to the kidney, bladder, or urethra. Hematuria may be due to the presence of a calculus in the kidney, ureter, or bladder. In rare instances it has been traced to the irritation of a highly concentrated urine, or of a pronounced case of the uric acid infarction of the newborn.

Hematuria is also a common and important symptom in many diseases in which there is a general tendency toward hemorrhage. The most important of these hemorrhagic diseases are hemorrhagic disease of the newborn, infantile scurvy, purpura, pernicious anemia and leukemia.

DIAGNOSIS.—To determine the source and cause of the hemorrhage is often quite difficult, and at times is impossible. When the blood comes from the bladder it may not be uniformly diffused through the urine, and small clots are common. In addition to this there are symptoms of disturbance of the bladder, such as tenesmus and frequent and perhaps interrupted micturition. In hemorrhage from the kidney the blood is diffused throughout the urine. The color may be red or brownish-red. The microscopic examination shows epithelium and casts from the kidney, many of which contain red blood corpuscles embedded in them, and are known as blood casts, and the renal elements are stained yellow and brown from long contact with the blood. There are also normal blood corpuscles, and others from which the hemoglobin has been washed out, which appear as pale rings, the biconcavity being lost.

The diagnosis of hematuria rests upon the detection in the urine by microscopic examination of red blood corpuscles, either in a normal condition, or decolorized.

TREATMENT.—The treatment of hematuria consists in that of the primary disease. The cause should be determined, if possible, and the indications for treatment followed. Rest in bed and a light diet until the urine is normal should be enforced.

HEMOGLOBINURIA.—In this condition blood pigment is found in the urine without red corpuscles. The urine is of a dark, reddish-brown color, and may be almost black. The condition is recognized by the fact that examination of the urinary sediment reveals few if any blood corpuscles, although usually pigment granules can be recognized. For ordinary clinical purposes the presence of blood pigment can best be recognized by the guaiac test (Vol. I, page 128), but for scientific certainty the spectroscope should be used.

Hemoglobinuria may be produced by certain poisons, such as carbolic acid or chlorate of potash. It is sometimes a manifestation of toxic nephritis. Infectious hemoglobinuria has been described under the Diseases of the Newborn.

There is a form known as *paroxysmal hemoglobinuria*, which, while very rare, is occasionally encountered in childhood. The cause is obscure. Some cases are probably of malarial origin; many others seem to be dependent upon over-exertion and exposure to cold, such as may come from chilled or wet feet, or from a cold-water plunge. From the etiological and pathogenetic point of view the disease is more properly classified with diseases of the blood.

SYMPTOMS.—With the onset of the attack there may be severe symptoms such as chills, fever, rapid and small pulse, and prostration. Again there may be hardly any subjective symptoms, the color of the urine alone being noticed. The attack lasts for a very variable period, and attacks occur at irregular intervals.

TREATMENT.—The treatment of this condition is very unsatisfactory. Prevention of exposure to cold is all that can be done.

GLYCOSURIA.—The persistent presence of sugar in the urine is symptomatic of diabetes mellitus. Transitory glycosuria is sometimes seen in early life. It is a rare symptom in some forms of toxic nephritis.

It has been generally stated in pediatric literature that there is a *dietetic* or *alimentary glycosuria*, often seen in infants who are taking an excess of sugar in the food. Grosz stated that sugar appears in the urine when more than a certain quantity is ingested. These statements I believe to be for the most part untrue. In some work carried out by Dr. Langley Porter and the writer at the Infants' Hospital, we were unable in any instance to cause the appearance of sugar in the urine, or an increase of already existing sugar in the urine, by giving an excess of sugar in the food. The effect of an excessive ingestion of sugar was always confined to the gastro-enteric tract, showing itself in increased fermentation. We often found slight traces of glucose in the urine in infants, but the presence or amount of sugar was entirely independent of the quantity of sugar ingested. We concluded that much of the literature of glycosuria in infancy is based on an error, through which creatinin, which reduces copper salts, and which is normally present in the infantile urine, was mistaken for sugar. The statement that glycosuria is often a symptom of severe gastro-intestinal disturbance, I believe to be based on the same error. In my opinion slight transitory glycosuria is occasionally seen in infants, but its cause is unknown. I cannot, however, deny the possibility of alimentary glycosuria in older children.

INDICANURIA.—Indican (indoxyl-potassium sulphate), is derived from indol, which is a product of protein putrefaction in the intestine. It may also be formed in other parts of the body where excessive protein decomposition is going on, such as occurs in extensive suppurative processes. It is not normally present in the urine of the breast-fed infant.

Indicanuria is a symptom of intestinal disease. Its value as a measure of intestinal putrefaction, and as a guide to treatment has, in my opinion, been much exaggerated in pediatric literature. The chief value of the indican test is in certain cases of indigestion with fermentation, in which it is difficult to decide whether the symptoms are due to a relative excess of protein or of carbohydrate.

ACETONURIA.—The significance of acetone and the acetone bodies in the urine has been thoroughly discussed under Acidosis. (Vol. I, page 402.)

CLASSIFICATION OF NEPHRITIS

There is no part of medicine in which the classification of diseased processes is attended by more difficulty than the various lesions of the kidney included under the term *nephritis.* Numerous attempts at classification have produced a most voluminous and confused terminology, because different aspects of the subject have been used as a basis for classification. Nephritis was first classified upon a purely clinical basis, and the various forms were differentiated mainly on a basis of the urinary findings. It is easy to understand how this came about. The clinical examination of the urine was the first step in the development of the numerous laboratory methods of diagnosis which now occupy so important a place in medicine. Then followed a period in which the prevailing tendency was to classify diseases on a basis of pathological anatomy. Still later, attention was turned to etiology, and the tendency of the present day is to regard classification on an etiological basis as preferable, whenever it can be attained. In nephritis there is a still more modern point of view, that of the pathological physiologist, which attempts to classify these diseases upon the basis of the functional capacity of the kidney as shown by special physiological tests.

It is extremely difficult for a clinical writer to present a classification which shall harmonize all these different points of view. In the diseases of the kidney there is a remarkable lack of correspondence between clinical types, anatomical lesions, etiological factors, and the functional disturbances revealed by special tests. A classification which shall assign its full value to all these different aspects of the subject is desirable, but impossible.

In order to clarify the subject as much as possible, I shall present a clinical, a pathological-histological, an etiological, and a pathological-physiological classification, each based on the most recent literature.

TABLE 69

Clinical Types of Nephritis

I. Acute cases, with albumin and often with blood in the urine, but without marked oliguria, edema, or uremic symptoms.
II. Acute cases with albumin and often with blood in the urine, with oliguria, edema, and sometimes with uremic symptoms.
III. Subacute or chronic cases with oliguria, a large amount of albumin, but no blood in the urine, and with renal edema, often terminating in uremia.
IV. Chronic cases with a normal quantity of urine or polyuria, with a small amount of albumin in the urine, but without edema, terminating in uremia or secondary cardiac disease.
V. Types in which fever and pyuria are the chief clinical manifestations.

TABLE 70

Pathological-Histological Types of Nephritis (Mallory)

I. Tubular nephritis. Degenerative lesions of the tubular epithelium, mainly confined to the convoluted tubules.
II. Capsular glomerulonephritis. Inflammatory reaction mainly in the capsular spaces of the glomeruli.
III. Intracapillary glomerulonephritis. Inflammatory reaction mainly within the capillaries of the glomerular tufts.
IV. Vascular nephritis. Primary lesion an arterio-sclerosis in the small arteries of the cortex.
V. Infectious nephritis. A diffuse inflammatory process which may go on to suppuration.
VI. Amyloid degeneration.

TABLE 71

Etiological Basis of Classification

I. Toxic nephritis—lesions due to soluble toxic substances reaching the kidneys through the blood.
II. Infections—
  a. Lesions due to bacteria reaching the kidneys through the blood.
  b. Lesions due to bacteria reaching the kidneys through the urine.

TABLE 72

Pathological-Physiological Basis of Classification

I. Vascular nephritis. Delayed lactose excretion.
II. Tubular nephritis. Delayed sodium chloride and potassium iodide excretion.
III. Uremic nephritis. Increase of non-protein nitrogen in the blood.

These specimen classifications will serve to illustrate the complexity of the subject. The various groups in the several schemes of classification do not correspond with one another, but overlap in such a way that it is impossible to present a scheme which will be consistent. Nevertheless the problem is somewhat more simple in children than in adults, because one great etiological and anatomical group is so rarely encountered in early life that it may be given a very minor place in clinical description. This is the group generally called "*vascular*," in which the primary lesions consist in arteriosclerotic changes in the vessels of the kidney. Arteriosclerosis, with its causes and lesions, plays little part in childhood.

The classification which I shall adopt in describing the diseases of the kidney usually included under the term nephritis, is based fundamentally upon etiology. The two principal causes of the lesions of the kidneys found in these diseases are (1) *the injury produced by soluble toxins circulating in the blood*, and (2) *the injury produced by the presence of bacteria in the renal tissues.* It is true that there is a certain amount of overlapping, both in the lesions, and in the functional disturbances produced by these two great etiological factors. Consequently there will of necessity be a certain element of inconsistency in the classification adopted here. I shall, however, endeavor to point out exactly where such inconsistency is encountered.

The primary subdivisions of the nephritides will be—

1. Toxic nephritis, or lesions produced by toxemias.
2. Infectious nephritis, or lesions produced by infection of the renal tissue.

Further subdivisions will be made upon a basis of pathological anatomy, because there is a certain degree of correspondence between anatomical and clinical types, which will make the clinical descriptions easier if made upon this basis.

The final classification, which will be used in clinical description, is the following:—

Toxic nephritis	Tubular nephritis Glomerular Nephritis Amyloid degeneration Chronic interstitial nephritis (rare)
Infectious nephritis	Suppurative nephritis Pyelo-nephritis

In order that the reader may grasp the relation of this classification to the various aspects of the subject, the following table is given, showing in a general way the amount of correspondence which exists between the clinical, pathological, etiological, and physiological aspects of the subject:

TABLE 73

Comparison of Types of Nephritis under Various Bases of Classification

BASIS OF DESCRIPTION	CLINICAL	PATHOLOGICAL-ANATOMICAL	ETIOLOGICAL	PATHOLOGICAL-PHYSIOLOGICAL
Tubular Nephritis	Acute cases without edema or uremia	Tubular	Toxic	Tubular
Glomerular Nephritis	1. Acute cases with edema 2. Subacute or chronic cases with edema	1. Capsular Glomerular 2. Intracapillary Glomerular	Toxic	Uremic
[Primary, Chronic, Interstitial Nephritis (rare)]	[Chronic cases with polyuria and without edema, and with raised blood pressure]	[Primary lesion arteriosclerosis]	[Toxic]	[Vascular]
Amyloid Infiltration	Chronic with edema	Amyloid	Toxic	
Suppurative Nephritis	"Surgical kidney"	Hematogenous Abscesses	Infectious (Hematogenous)	
Pyelonephritis	Pyelitis	Pyelonephritis	Infectious (Through urine or blood)	

IV. LESIONS PRODUCED BY TOXEMIAS—TOXIC NEPHRITIS

TUBULAR NEPHRITIS

(Acute degenerative nephritis) (Acute degeneration of the kidneys)

Clinical Type.—Acute cases without edema, uremic symptoms, or marked oliguria.

Anatomical Type.—Degeneration of the tubular epithelium with inflammatory reaction in the tubules or interstitial tissue.

Etiological Type.—Lesions caused by bacterial or chemical toxins.

Physiological Type.—Delayed excretion of sodium chloride and potassium iodide.

The term *nephritis* is by many writers considered not strictly applicable to this disease of the kidneys, because the lesions are not primarily of an inflammatory character. For this reason many modern writers prefer the term *nephrosis* in describing diseases characterized by non-inflammatory renal lesions. The term which best describes the etiology and pathological anatomy is acute or toxic degeneration of the kidneys. The term *tubular nephritis* is retained here because while a degeneration of the renal epithelium is the primary lesion, it is quickly followed by an inflammatory reaction. Furthermore nephritis is the term most frequently employed by the pathological anatomist in describing the lesions found at autopsy.

ETIOLOGY.—The lesions are produced by the action on the renal epithelium of soluble toxins circulating in the blood. Three varieties of toxins may cause an acute degeneration of the renal epithelium. They are the following:

1. Bacterial toxins.
2. Endogenous chemical toxins.
3. Exogenous chemical toxins.

Bacterial toxins are the commonest cause of tubular nephritis in children. The condition is found to a varying degree in all autopsies upon children dying from acute infectious diseases. It is most marked in cases of scarlet fever, diphtheria, and pneumonia. Any infection may produce the lesions, even a slight infection of the upper respiratory tract. Tubular nephritis is the cause of the so-called febrile albuminuria seen in so many acute infections.

The endogenous chemical toxins which may produce acute degeneration of the renal epithelium may originate in the intestinal canal, or in the tissues of the body. The toxins absorbed from the intestinal canal are the result of abnormal bacterial fermentation of the food substances in the intestine. This explains the evidences of renal irritation sometimes seen in non-infectious gastro-intestinal disease. There is evidence that in certain diseased conditions toxic products are formed as a result of a disturbance of the general metabolism. This explains the evidences of renal irritation sometimes seen in nutritional disorders without gastro-enteric symptoms.

The exogenous chemical toxins are various drugs. Among these irritating drugs are turpentine, cantharides, corrosive sublimate, bicarbonate of potash, arsenic, lead, salicylic acid, and potassium chlorate.

PATHOLOGICAL ANATOMY.—Macroscopically the condition is that commonly described as cloudy swelling. The kidneys are slightly larger, paler and softer than normal. The cortex is some what wider than normal. There is seldom much congestion of the vessels. Microscopically the lesions are seen to be confined mainly to the convoluted tubules, and may be diffuse and extensive, or confined to scattered foci. There may be extensive necrosis of the epithelium lining the convoluted tubules, or necrosis may not be so marked, the cells showing hydropic and hyaline changes. The tubules may be filled with desquamated epithelium. The inflammatory reaction is usually mainly shown by the presence in the tubules of serum, fibrin, and polymorphonuclear and endothelial leukocytes. There is often some edema and lymphocytic infiltration of the interstitial tissue. In some cases this interstitial infiltration is so marked, that acute interstitial nephritis has been described as a separate disease. It is probable that of the cases described by the pathologists as acute interstitial nephritis, some belong in this group, the primary lesion being an injury to the tubular epithelium, and the cellular infiltration of the interstitial tissue being a secondary inflammatory reaction. Other cases showing the same interstitial lesions probably belong in the group in which the inflammatory reaction is cased by the actual presence of bacteria in the interstitial tissue

SYMPTOMS.—In acute tubular nephritis there are no symptoms other than those of the primary disease.

THE URINE.—In typical cases the urine is clear, acid, and its color is often normal. If the irritation is secondary to a febrile infection, the urine is generally high-colored, concentrated, and slightly diminished in quantity. If the cause is not febrile, the urine is often either normal in quantity or dilute. The specific gravity is generally increased. The solids are either normal or slightly diminished. There is a slight sediment, with a slight trace of albumin. Microscopic examination shows the presence of renal epithelium and blood corpuscles; the latter, however, are not ordinarily in sufficient number to color the urine. There are also leukocytes, and hyaline and fine granular casts with an occasional epithelial cast and blood cast.

In the severer forms of irritation with extensive necrosis of the renal epithelium, the albumin may be present in larger amounts. There is sometimes sufficient hematuria to give the urine a slight smoky or reddish color.

DIAGNOSIS.—The only other acute forms of nephritis commonly encountered in early life are acute glomerulonephritis, and acute pyelonephritis. The latter condition is recognized by the purulent character of the sediment, the greater proportion of the cellular elements being polymorphonuclear leukocytes. The differential diagnosis between acute tubular nephritis and acute glomerulonephritis cannot be made on the basis of the quantity of albumin and character of the sediment found in the urine. It is true that in acute glomerulonephritis the quantity of albumin, quantity of blood in the sediment, and number of casts and renal elements in the sediment are usually larger than in acute tubular nephritis. Nevertheless severe cases of tubular nephritis are sometimes encountered with considerable albumin, blood, and renal elements, while there are mild cases of glomerulonephritis in which the urinary findings are exactly like those seen in acute degeneration.

In the recognition of tubular nephritis, the three most important points in differential diagnosis are the following: (1) *The absence of edema;* (2) *the absence of a marked diminution in the quantity of urine excreted;* (3) *the absence of any uremic, local, or general constitutional symptoms which can be attributed to the process in the kidneys.*

These points will always serve to distinguish acute tubular nephritis from average and typical cases of acute glomerulonephritis. There are certain atypical cases, without edema and without marked oliguria, in which the lesions of glomerulonephritis have been found postmortem. Some of these are, from the pathological-anatomical point of view, intermediate types. They usually have constitutional symptoms attributable to the nephritis, particularly a rapidly developing anemia. If there are no general constitutional symptoms. the only possible diagnosis at first is tubular nephritis. This is confirmed if the evidences of nephritis disappear rapidly with the disappearance of the primary cause, but if they tend to persist and run a long course, glomerulonephritis should be suspected.

PROGNOSIS.—The prognosis of acute tubular nephritis is favorable. When the lesions are confined to the convoluted tubules, regeneration is rapid and complete, and the products of inflammatory reaction are absorbed without the proliferation of fibroblasts and the formation of fibrous tissue. Chronic fibrous changes only take place in diffuse inflammation involving all the renal elements, and this is not seen in tubular nephritis.

TREATMENT.—No special treatment is required for the disease of the kidneys, the general treatment being that of the primary condition. The diet in acute infectious disease should always be largely liquid. When signs of toxic irritation of the kidneys occur, the physician should always make sure that a sufficient quantity of fluid is taken daily. It is also well in cases of acute degenerative nephritis accompanying the acute infections, to have the daily quantity of urine measured and recorded. This is useful in making sure that sufficient fluid is being taken, and also in recognizing the development of a more serious form of nephritis.

GLOMERULONEPHRITIS

(Diffuse Nephritis) (Parenchymatous Nephritis)
(Exudative Nephritis)

Clinical Type.—Acute or chronic cases with oliguria, edema, and often with uremic symptoms.
Anatomical Type.—Capsular or intracapillary glomerulonephritis.
Etiological Type.—Bacterial Toxins.
Physiological Type.—Increase of non-protein nitrogen in the blood.

ETIOLOGY.—The only important cause of this form of nephritis is the toxins produced by bacteria. The great majority of cases encountered in childhood are secondary to some recognized acute infectious disease. The most common and well-known infection with which diffuse nephritis is associated is *scarlet fever*. Another very common cause of nephritis in children is *tonsillitis*. Glomerulonephritis is a fairly common complication in *severe septic conditions* caused by the streptococcus group of microörganisms. The disease is occasionally seen as a complication or sequela of diphtheria, measles, smallpox, meningitis, pneumonia, typhoid fever, erysipelas, malaria, empyema, influenza, and infectious diarrhea. In all of these conditions, however, tubular nephritis is the usual manifestation of the action of the toxin upon the kidneys, glomerulonephritis being exceptional.

I must here point out one of the inconsistencies which necessarily attends an attempt to classify the nephritides upon an etiological basis. Glomerulonephritis is classified as a disease in which the lesions are produced by a toxemia, whereas the typical lesions produced by the presence of the bacteria themselves which reach the renal tissues through the blood stream are classified under the heading of suppurative nephritis. This distinction is made necessary by the anatomical and clinical differences between the two conditions. Nevertheless, it is probable that the characteristic lesions of glomerulonephritis can be produced not only by bacterial toxins brought to the renal tissues by the circulating blood, but also by the toxins formed by bacteria actually present in the renal tissues, these bacteria being of hematogenous origin. There is no doubt that bacterial toxins can produce the characteristic lesions of this disease without the presence of bacteria in the tissues. This has been demonstrated in the case of streptococcus infection, for in cases dying of septic sore throat, which is a streptococcus infection, the lesions of glomerulonephritis are found in the kidneys without the presence of streptococci. The acute nephritis which complicates cases of ordinary tonsillitis is probably similarly caused by the action of streptococcus toxins. Indeed, there is at present accumulating much evidence that the lesions of glomerulonephritis are particularly associated with streptococcus infection. Nevertheless there is also evidence that the same lesions may be caused by the presence of the actual streptococci diffusely scattered in the renal tissue, and in the cases of nephritis complicating septicemias or severe septic infections, such a localization of the organisms is highly probable. Such cases, from the etiological point of view, should be classified with acute suppurative nephritis, but from the point of view of pathological anatomy and clinical symptoms, they belong with the diffuse inflammation of the kidney in which the chief lesions are in the glomeruli.

Whether the glomerulonephritis which follows scarlet fever is due to the secondary streptococcus infection which so often occurs in that disease, the renal lesions being due to streptococcus toxins either brought by the blood, or formed in the kidneys, or whether, as seems more likely, the renal lesions are due to the action of the unknown virus of scarlet fever upon the kidneys, is unknown.

In the other infections, in which glomerulonephritis is comparatively uncommon, the same uncertainty exists as to the exact pathogenesis of the renal lesions. There is certainly evidence that they *can* be caused by the presence of toxins only. Whether they *are* always so caused is not known. They may be due to the toxins produced by the specific virus, by the presence of the specific virus itself, by the toxins absorbed from a complicating streptococcus infection, or by the presence of secondary streptococcus invaders. It is probable that the pathogenesis of the disease is variable, and that some of the irregular and atypical clinical types are dependent upon these variations.

A certain number of cases of glomerulonephritis are encountered in early life, in which the disease is *apparently primary*. In explanation of such cases there are two possibilities. The first is that the nephritis complicates a latent infection, that is, an infection which does not manifest itself clinically. That such infections exist is becoming a well-known fact in medicine. The bearing of latent focal infection upon certain general symptoms has attracted much attention of late. It is the members of the streptococcus group which are particularly associated with infections of this kind, and it is easily conceivable that streptococci or streptococcus toxins derived from such foci might be the cause of the kidney lesions. The other possibility in primary nephritis is suggested by recently studied peculiarities of the streptococcus group of microörganisms. Rosenow's work suggesting an acquired selective affinity of streptococci for certain tissues suggests that at times nephritis may be due to such an affinity, and under this possibility some cases of nephritis may represent a true primary streptococcus infection of the kidneys.

Exposure to cold is always mentioned in textbooks as an etiological factor in acute nephritis. Bartels calls it an etiological scapegoat as regards nephritis, and I am decidedly of this opinion. A history of exposure to cold preceding the development of nephritis is more common in adults than in children. One explanation is that the exposure produces a general disturbance of metabolism with the formation of toxic products which irritate the kidney. This explanation seems to me highly improbable. All the available evidence, including the argument from analogy, points toward the view that the chief cause of the lesions of glomerulonephritis is bacterial toxins. That they may be produced experimentally by exogenous chemical toxins does not prove, in default of other evidence, that they are caused by endogenous chemical toxins. The influence of cold in predisposing toward infection, or in increasing the activity of the bacteria in a focal or latent infection, is well-known, and this seems a more plausible theory than an obscure disturbance of metabolism.

Glomerulonephritis, at any rate in its acute stage, is more common in childhood than in adult life. This is undoubtedly due to the greater frequency of the acute specific infections in early life. It is more common in later childhood than in infancy, but may occur at any age of childhood. If the cases following scarlet fever be excluded, it occurs with about equal frequency in infancy and later childhood. The type in which the disease is apparently primary is chiefly seen in infancy. This is probably due to the difficulty in recognizing some infectious processes in infancy. Careful examination has shown that many supposedly primary cases in infants are due to tonsillitis.

PATHOLOGICAL ANATOMY.—Macroscopically in severe cases the kidneys are enlarged, soft and edematous, and the capsule is not adherent. The cortex is wider than normal, and may be pale, congested, or mottled with small hemorrhages. In many cases the congestion of the vessels causes the pyramids to stand out prominently in contrast with the paler cortex.

Microscopically all the renal elements, glomeruli, tubules and interstitial tissue, show more or less evidence of an inflammatory process. For this reason the term *diffuse nephritis* is frequently employed in the description of this disease. The kidney is composed of a number of units, each unit consisting of an afferent vessel, a knot of capillaries in the glomerulus, the blind end of the tubule applied to this knot of capillaries as a capsule, and the tubule. If any one of these elements be injured and destroyed, the others will atrophy. It is the glomerulus, where blood vessels and epithelium are most closely associated, that suffers most from injurious toxic agencies. Consequently the primary and typical lesion is found in the glomerulus, although the inflammatory reaction is not confined to the glomerulus, but involves all parts of the renal unit. The term *diffuse nephritis* is perhaps more descriptive than any other of the appearances seen in many cases. On the other hand, the most constant and typical lesions are seen in the glomeruli. The term *glomerulonephritis* is chosen, because it is the one usually employed by the pathologist in describing the microscopic lesions found post-mortem. It is to be remembered, however, that as used here, the terms glomerulonephritis and diffuse nephritis are synonymous.

In the description of the lesions found in the glomeruli, the terms capsular, intracapillary, exudative, and proliferative are used. In the description of other aspects of the diffuse inflammation, the terms acute interstitial and hemorrhagic are encountered.

In *capsular glomerulonephritis* the chief inflammatory reaction to toxins takes place within the capsular space. When the reaction consists principally in the exudation of serum containing albumin, and in the emigration of polymorphonuclear and endothelial leukocytes, occasionally with fibrin formation, the process is called *exudative.* When the reaction consists chiefly in a proliferation of the epithelial cells lining the capsular space, the process is called *proliferative.* When hemorrhage occurs, the term *acute hemorrhagic nephritis* is sometimes employed by the pathologist without reference to the real underlying lesion.

In *intracapillary glomerulonephritis* the chief inflammatory reaction to toxins takes place within the capillaries of the glomerular tuft. In the most acute cases the reaction is shown mainly by fibrin formation. In less acute cases, the process consists chiefly in accumulation of polymorphonuclear leukocytes within the capillaries, and in still slower processes the accumulated cells are chiefly endothelial leukocytes. The exudate causes plugging of the capillaries and distention of the tuft. There may be an associated capsular inflammation.

The tubules usually become filled with the products of the inflammatory reaction in the glomeruli. The toxin also usually causes more or less degeneration of the tubular epithelium, similar to that seen in tubular nephritis where the tubules alone are involved. The injury to the tubules may cause an inflammatory reaction in the adjacent interstitial tissue, shown by exudation of serum and infiltration by lymphocytes. When this reaction is marked, the term *acute interstitial nephritis* is sometimes employed, but this term describes a lesion which may be present both in tubular and in glomerular nephritis.

In some cases the accumulated products of the inflammatory reaction may disappear entirely. In many cases, probably in the majority, a return to a normal condition is prevented, owing to the formation of fibrin. Fibrin stimulates the fibroblasts in the tuft or capsule to proliferation and organization of the exudate by the formation of fibrous tissue. The result is a gradual more or less complete destruction of the glomerulus, by a sclerotic process which destroys the renal elements. When fibroblastic proliferative changes are active, the lesion is often described by the pathologist as *subacute* glomerulonephritis. When sclerosis has occurred, the process is often designated as *chronic.*

The inflammatory reaction in the interstitial tissue may also cause fibroblastic activity, and the formation of fibrous tissue. This may lead to a fibrous contraction of the entire kidney. Some pathologists, struck by the prominent character of the interstitial lesions, and overlooking the fact that the primary lesion is represented by the sclerosed glomeruli, have described such cases as *chronic interstitial nephritis.* This has led to the confusing of such cases with the primary chronic interstitial nephritis (vascular nephritis) so common in adults. German writers have always clearly differentiated the two conditions, calling chronic vascular nephritis *primary contracted kidney*, and the end stage of glomerulonephritis with interstitial lesions, *secondary contracted kidney.*

In very severe cases of acute glomerulonephritis, particularly in the type characterized by intracapillary inflammation, the blocking of the capillaries may be so complete that death may occur in a few days from uremia. Capsular types are, however, commoner in early life.

SYMPTOMS.—The three characteristic symptoms of glomerulonephritis are the following: (1) *Anomalies of the urinary secretion;* (2) *edema;* (3) *toxic or "uremic" symptoms.* The anomalies of the urinary secretion are the only localizing manifestation pointing directly at the kidney as the site of the lesions. They are due both to the functional incapacity of the kidneys caused by the lesions, and to the presence in the urine of the products of the inflammatory reaction itself. The functional damage is shown by a diminution of the excretory power of the kidney. There is a diminution in the quantity of urine excreted from damage to the power of eliminating water, and also certain changes in the chemistry of the urine due to a deficiency in the power of eliminating salts and nitrogenous waste products. The products of the inflammatory reaction found in the urine are albumin, blood corpuscles, leukocytes, renal epithelium, and casts due to hyaline epithelial degeneration.

The pathogenesis of the *edema* seen in glomerulonephritis has formed the subject of much discussion. The prevailing theory is that the edema is due to retention of water and salts in the blood and body tissues, plus a toxic action on the walls of the blood vessels.

The *uremic symptoms* are due to the retention in the blood of the non-protein nitrogenous substances which are the waste products of protein metabolism.

From the clinical point of view, cases of glomerulonephritis are either *acute* or *chronic*. In a general way there is a correspondence between these clinical types and the pathological changes in the kidneys. The acute cases are those in which the action of the bacterial toxin causes an acute inflammatory reaction, usually of an exudative type, but occasionally with toxins of less strength, of a proliferative type. The chronic cases represent the results of the inflammation as seen in fibrous tissue formation and destruction of renal elements. The lesions are often in a stage described by the pathologist as *subacute*, but the most convenient clinical division is into acute and chronic.

ACUTE GLOMERULONEPHRITIS

(Acute Diffuse Nephritis) (Acute Exudative Nephritis)
(Acute Bright's Disease)

TYPICAL FORM.—The most typical form of acute glomerulonephritis is that which occurs as a sequela of scarlet fever. Cases which occur as a complication of tonsillitis in infancy, or occasionally in other acute infections, present a very similar clinical picture, but are apt to be somewhat less severe. Usually the first symptom noted is either *edema*, or *hematuria*. Edema shows itself as a puffiness of the face, particularly about the eyes. The edema may be confined to the face, but usually extends to other parts of the body, the feet and legs becoming involved after the face, then the scrotum, and then the lumbar region of the back. The edema may increase to

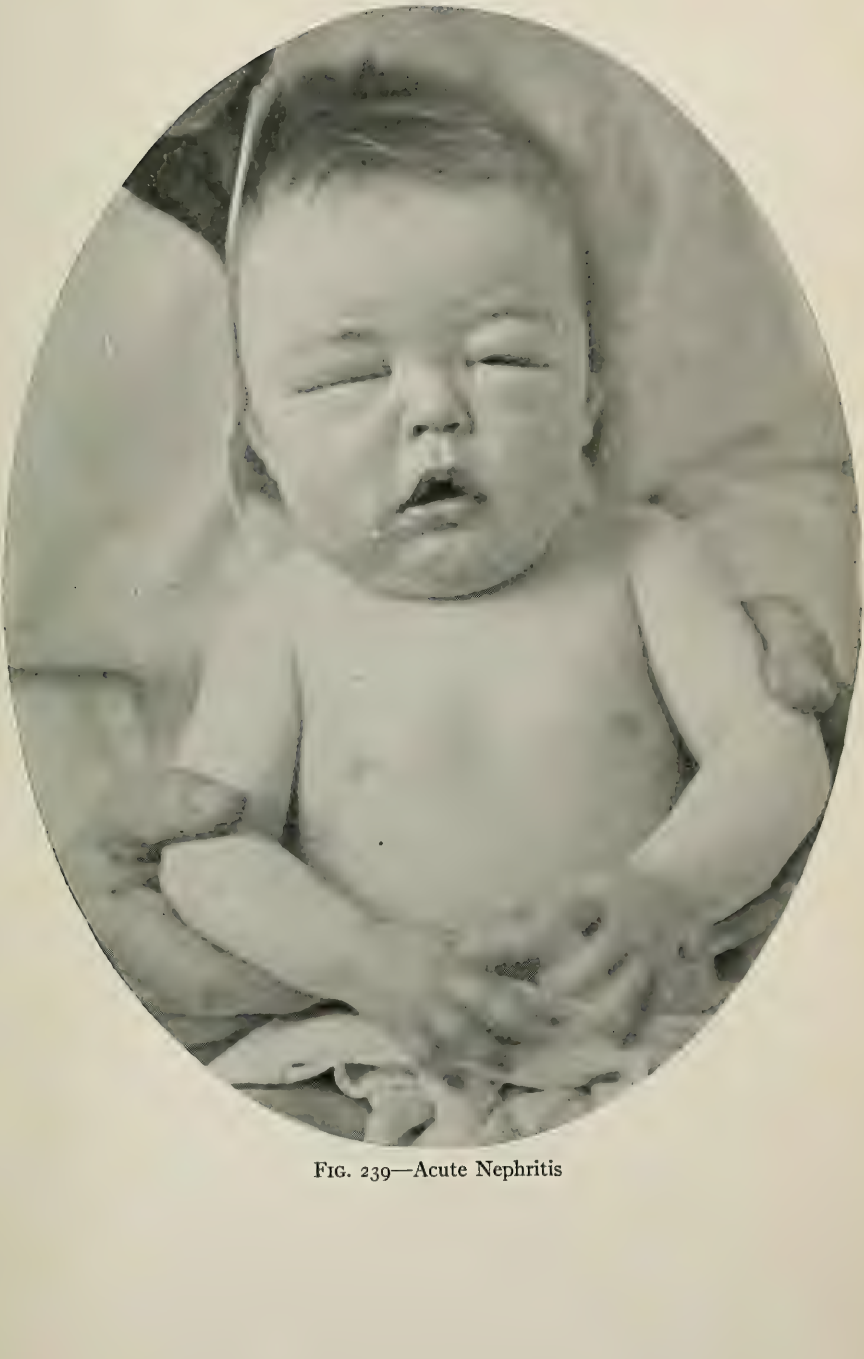


FIG. 239—Acute Nephritis

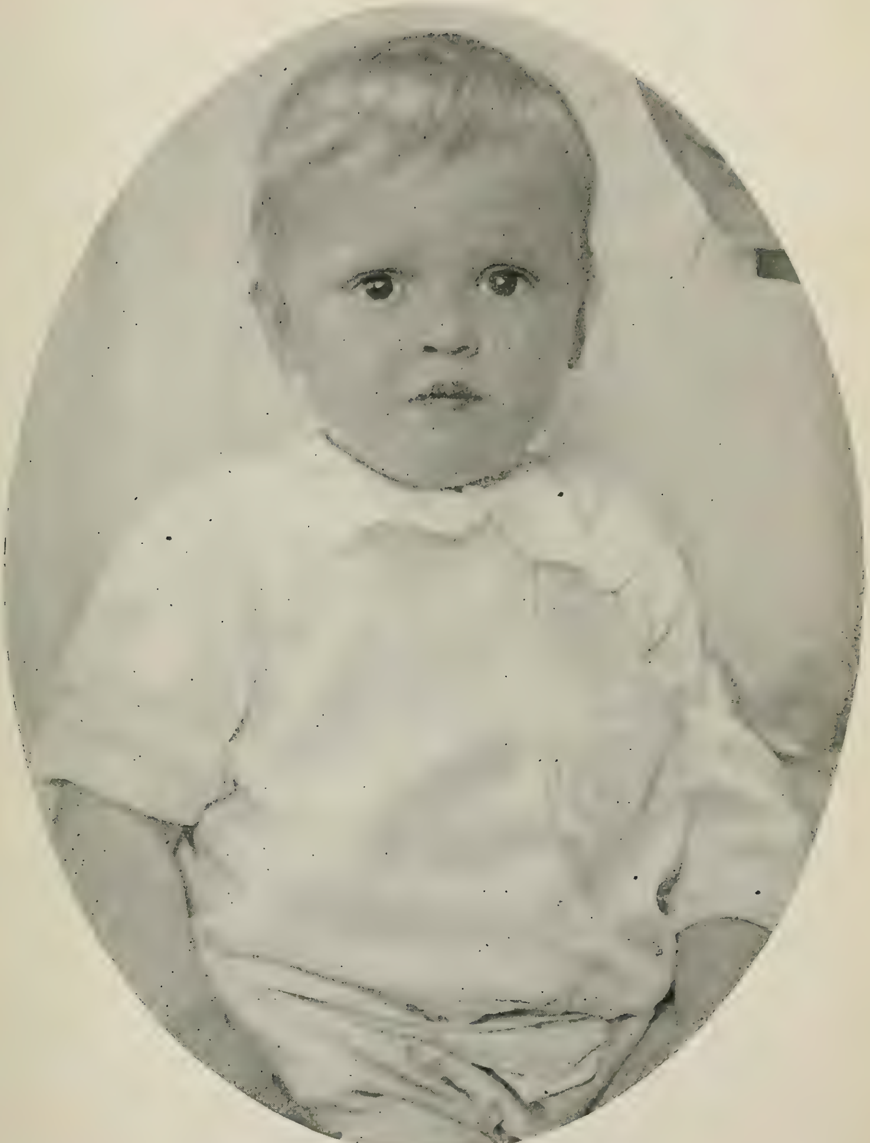


FIG. 240—Acute Nephritis after recovery

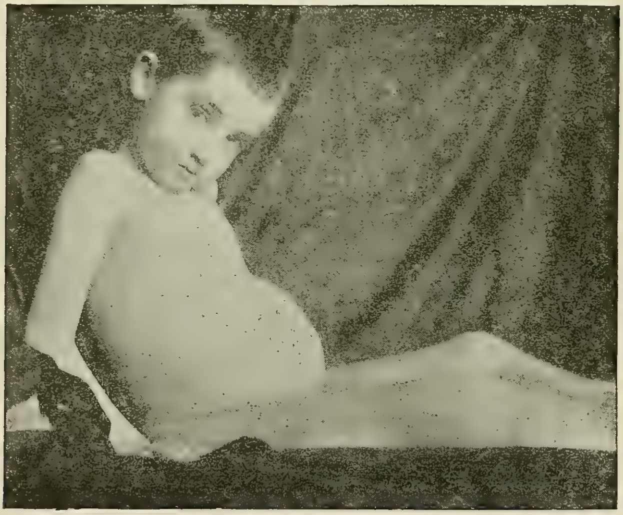
a general anasarca. There may be transudation into the serous cavities, most often into the peritoneal cavity, at times into the pleural cavities, rarely into the pericardial cavity. Dropsy of the serous cavities is shown by the usual signs of ascites, pleural effusion, or pericardial effusion. Often in infants the first sign of approaching edema is a sudden inexplicable gain in weight. A rapidly developing anemia is a very constant and characteristic symptom.

With the edema, uremic symptoms may develop. The most common uremic symptom in children is vomiting. In some cases there is headache and dimness of vision. In a few cases, with very scanty or suppressed urine, there are even convulsions. The temperature is usually moderately raised, from 100 to 101.5° F., but in severe cases it may go much higher. In some cases uremic symptoms may be more marked than the edema at the onset. In such cases the onset is abrupt, with vomiting, headache, fever, scanty urine, and occasionally convulsions. These cases, however, are the exceptions rather than the rule in early life. As compared with acute nephritis in adults, in typical cases in children edema is more prominent as a clinical manifestation, and uremic symptoms are less prominent. The typical onset is with edema, and most cases run their entire course without uremic symptoms. On the other hand there are occasional cases in which marked uremic symptoms, suppression of urine, and uremic convulsions and coma, develop rapidly.

The urine, as a rule, is very much diminished in quantity. At times there may be complete suppression, the anuria lasting up to twenty-four hours or even longer. It is under such circumstances that uremic symptoms are more likely to be present and severe. The color is dark, smoky or blood-red, and this may be the first symptom noted by the child's mother. The reaction is usually acid, rarely alkaline from the presence of blood. The specific gravity is low or high; it may be high from the presence of albumin. The normal solids are diminished both absolutely and relatively, especially the chlorine and urea. The albumin varies in amount, being usually from one-fourth to one-half of one per cent., but it may be much less or much more. The sediment is abundant and is of a dark brown color. There are large numbers of normal and decolorized blood corpuscles, brown granular detritus, renal cells and leukocytes. Brown granular, epithelial, blood, fibrinous, and even finely granular and hyaline casts are seen in abundance.

In cases which run a favorable course, and which constitute a majority of the cases of acute nephritis seen in early life, the duration of active symptoms is from one to three weeks. Improvement as shown by diminution of edema, lessening of uremic symptoms, and increase in the quantity of urine, usually begins in from three days to a week or ten days. In this stage the quantity of urine and solids excreted begins to increase, the albumin to diminish, and the sediment to become more dilute, with the same characteristics, but with the addition of fatty renal cells, and fatty casts. The temperature falls to the normal, and the dropsy gradually subsides and disappears. During convalescence there is a marked increase in the quantity of urine, which gradually loses its dark and smoky color and becomes pale, faintly acid, of low specific gravity, and with only a trace of albumin. The solids are nearly normal in absolute amount, but relatively are diminished, owing to the increased amount of urine. The sediment becomes slight in quantity, colorless, and shows a great diminution in the number of renal elements. The urine may become normal in a few weeks. In other cases a small amount of albumin and a few casts and epithelial cells may persist in the urine for from several months to several years, and still complete recovery may occur. In still other cases the persistence of albumin and casts is a sign that the damage done to the kidney is more serious and permanent, and that lesions are present which will eventually lead to the clinical manifestations of chronic nephritis. It is a possibility that albumin and casts may disappear completely from the urine in convalescence, and that nevertheless the damage may be sufficient to lead to a chronic nephritis, which may manifest itself at any future time.

FIG. 241



Acute glomerular nephritis, following scarlet fever

Severe cases, typical except for the grave character of the symptoms, are sometimes seen in children, especially after scarlet fever.

They are, however, comparatively uncommon. The onset is accompanied by high fever, pain in the lumbar region, and a rapid full pulse of high tension. The urinary secretion is very scanty, or entirely suppressed. Vomiting, with restlessness or apathy soon develops, and there may be a diarrhea. Edema is present but is less marked than the uremic symptoms. In some cases there is headache, dimness of vision, and rapid deep breathing. Stupor deepening into coma, or convulsions may occur. Death may occur, but in typical cases even in this severe type, the urinary secretion is usually reëstablished, even after as long as thirty-six hours of anuria, and recovery follows. In cases with suspected suppression of urine, the physician should make sure that the anuria is really due to suppression and not to retention. Percussion of the bladder and catheterization will settle this point.

ATYPICAL FORMS.—There are certain comparatively rare cases seen in early life in which the clinical manifestations differ so much from those seen in typical cases of glomerulonephritis, that the diagnosis is extremely difficult. Most of these atypical cases are seen in infants. The disease appears clinically as a primary nephritis, that is, there is no definite evidence of an infectious process localized elsewhere. The onset is usually abrupt with high fever and vomiting. The disease runs a course of from one to three weeks, the chief clinical manifestation being high irregular fever. Edema is absent, except possibly toward the end. Vomiting and diarrhea are usually present, but are not marked, and seem to be due to indigestion rather than to uremia. The mental condition is dull and apathetic, but there is rarely complete coma even when a fatal ending is approaching. Restlessness and muscular twitching are usually prominent. Anemia is marked. The urine is not so markedly diminished in quantity as in the typical form of the disease, until near the end. There is less albumin than in typical cases, and less blood, the sediment consisting in pus cells, renal cells, a moderate number of red blood corpuscles, and a few hyaline and finely granular casts.

The absence of edema and of a marked diminution in the amount of urine, and the urinary findings, suggest a tubular rather than a glomerular nephritis. The clinical picture is that of severe infection of obscure localization, with constitutional symptoms and the urine of a tubular nephritis. Nevertheless in a certain number of cases of this type occurring in infants, glomerulonephritis has been found at autopsy.

There are also rare atypical cases seen in older children, which are clinically suggestive of the tubular form of nephritis. They differ from typical cases of glomerulonephritis in the absence of edema, uremic symptoms, or marked diminution in the quantity of urine. They differ from typical cases of tubular nephritis in that they are apparently primary. They are not so fatal as the atypical cases seen in infants, and our knowledge that the disease represents the glomerular type of nephritis is based on the fact that some of these cases later developed the typical clinical picture of chronic glomerulonephritis.

CHRONIC GLOMERULONEPHRITIS

(Subacute Glomerulonephritis) (Chronic Parenchymatous Nephritis)
(Chronic Diffuse Nephritis)

The majority of cases of this disease represent the further progress of the pathological changes caused by an acute attack. The symptoms may follow immediately after the acute attack, but there is more often an interval of months or years during which the patient is believed to have entirely recovered, or during which the only clinical manifestation is the persistence of a small amount of albumin and a few casts in the urine. In some cases, the disease appears to be chronic from the start. The etiology of these cases is very obscure. It is possible that in some acute infection the manifestations of acute nephritis were so slight, that they were either overlooked, or were attributed to the tubular form of the disease. There is aslo the possibility that the toxin may give rise to chronic lesions without the clinical manifestations of acute inflammation.

The onset in cases which are apparently chronic from the start, or which appear after an interval of freedom from clinical symptoms, is usually insidious. In many cases the only notable symptom is the gradual development of edema. In other cases the onset is with loss of appetite, indigestion, and occasional vomiting. Edema is always present. The face becomes pallid and pasty, then puffiness is noted about the eyes. Gradually the whole face becomes involved, then dropsy appears in the lower extremities. In mild cases this is the extent of the edema. More often the dropsy extends to the scrotum and lumbar region, and finally a general anasarca develops. Ascites is fairly common, and the quantity of fluid may be very large. Hydrothorax is sometimes seen but is less common than ascites. Hydropericardium is seen at times.

In many cases, the dropsy is the principal clinical manifestation of the disease. In some cases, nervous symptoms of a uremic character are pronounced. There may be headache, restlessness, sleeplessness, and neuralgic pains in the extremities. Vomiting may occur occasionally, or at times may be frequent and severe. Palpitation of the heart and dyspnea are often seen, and the patient may be easily exhausted by slight exertion. Anemia is usually very marked. Epistaxis may occur.

The disease is characterized by periods of activity and quiescence.

In the active stages the edema is increased, the quantity of urine is very small, and uremic symptoms may threaten. The urine is high-colored, strongly acid, of high specific gravity, and the solids are absolutely much diminished, although relatively they may be increased. The twenty-four hour amount of urine is markedly diminished. The amount of albumin is usually very large, averaging from one-half to one per cent, although it is sometimes much higher. The sediment consists of many hyaline, granular, and fatty casts, some of which have fatty renal cells adherent. These fatty degenerated cells are also found free. In advanced stages waxy casts will be met with and are usually of bad prognosis. Signs of acute irritation of the kidney are often present and are indicated by the presence of blood elements.

During the quiescent stage the dropsy becomes absorbed, and the edema may entirely disappear. This change is associated with a marked increase in the quantity of urine, which may become normal or even in excess of the normal limit. As a result, the urine becomes pale, of a low specific gravity, and with solids which are both relatively and absolutely diminished. The albumin may remain large in quantity, or may become small. The sediment is the same as in the active stage, but the renal elements are less numerous.

The duration of chronic glomerulonephritis is very variable, depending on the hygienic surroundings of the patient, on the treatment, and on the occurrence and severity of intercurrent infections. The duration is rarely shorter than two years, and may last for many years. There may be long periods of quiescence, during which there is no edema, no toxic symptoms, and no marked diminution of the quantity of urine excreted. The general health may improve greatly during these periods, and the only sign of the disease is the persistence in the urine of a variable amount of albumin and of a few renal elements. These periods are eventually interrupted, either by the gradual appearance of a more active stage, or by the sudden appearance of an acute exacerbation. When the active stage comes on gradually, there is usually no apparent cause for the reappearance of symptoms. Edema reappears, the quantity of urine excreted falls, the quantity of albumin and the number of renal elements rise. There may then be a long period of persistence of active symptoms. In an acute exacerbation, edema or uremic symptoms come on more suddenly, the quantity of urine falls more rapidly, and blood reappears in the urine, the sediment resembling that of the acute form of the disease. These acute exacerbations are often precipitated by some infection.

Death may occur from edema of the lungs, acute uremia with convulsions and coma, or from some intercurrent infection such as pneumonia, pleurisy, pericarditis or endocarditis.

DIAGNOSIS.—The conditions to be considered in the differential diagnosis of glomerulonephritis are the following: Acute tubular nephritis; pyelo-nephritis; suppurative nephritis; hematuria from other causes; acute or chronic endo- or pericarditis with chronic passive congestion; toxic edema; orthostatic albuminuria; simple retention of urine; the anasarca and anemia seen in the primary anemias; chronic interstitial nephritis.

Acute Tubular Nephritis.—The essential points in the differentiation of acute glomerulonephritis from acute tubular nephritis are the presence in the former of *edema*, of *marked diminution in the daily quantity of urine*, and of *uremic symptoms.* If any one of these three symptoms are present, the nephritis is of the glomerular form. In the rare atypical cases of glomerulonephritis seen in infants, without edema or marked diminution in the quantity of urine, the diagnosis is often very difficult. In such cases glomerulonephritis is suspected when the acute inflammation of the kidneys is apparently primary, and accompanied by high fever and marked nervous symptoms.

Pyelonephritis.—In this condition the principal differentiating point is the character of the urinary sediment, which consists chiefly in pus cells. There is no marked diminution in the quantity of urine, no edema, blood is not present in an amount sufficient to be noted macroscopically, and casts are few in the sediment.

Suppurative Nephritis.—This condition is rare in children, and there may be no recognizable symptoms. When such symptoms are present, they are usually localizing in character, pointing toward the kidney, such as pain and tenderness in the region of the kidney. The urine may contain albumin and casts, but there is no edema, no marked diminution in the quantity of urine, and no uremic symptoms.

Hematuria from Causes Other than Nephritis.—The presence of blood in the urine always suggests nephritis, and the blood causes the urine to give a positive reaction for albumin. In hematuria from other causes the essential point in the exclusion of acute nephritis is the *absence of casts.* There is also no edema, no diminution in the quantity of urine secreted, and no uremic symptoms.

Cardiac Disease.—The passive congestion seen in acute endo- or pericarditis, and in uncompensated chronic cardiac disease, causes certain symptoms suggestive of nephritis, particularly edema, a diminution in the quantity of urine excreted, and the presence in the urine of albumin and casts. The recognition of a definite cardiac lesion, with the dyspnea, enlarged tender liver, and other symptoms of cardiac weakness, is a great aid in diagnosis. The mere finding of an organic cardiac murmur is not enough. The physician must be convinced from the cardiac symptoms that there is sufficient weakness of the heart to cause a passive congestion of the kidneys. There are other helpful points in differential diagnosis. The quantity of albumin and number of tube casts and cellular elements are less in cardiac disease than in glomerulonephritis, and there is not so much diminution in the urinary output. Edema of cardiac origin usually begins in the lower extremities, while the edema of nephritis usually begins in the face. The so-called "cardio-renal" cases in which dilatation and weakness of the heart are secondary to nephritis, are not seen in early life, and cardiac symptoms are usually clearly due to primary disease of the heart. In uncomplicated cases of nephritis, both in the acute and chronic stage, the examination of the heart is usually normal.

Toxic Edema.—The typical "angioneurotic" edema is easily distinguished by the localized character of the swelling. There is, however, a form of toxic edema which is quite common in infants as an accompaniment of chronic gastro-intestinal disease. In these cases the edema often begins in the face, and in its development resembles the edema of glomerulonephritis in every way. The differentiating point is the entire absence of albumin and renal elements in the urine. The frequency of this toxic edema of infancy is often forgotten, and I have often been called to see cases in which the physician was at a loss to comprehend why the urine should be normal in an otherwise apparently typical case of nephritis.

Orthostatic Albuminuria.—This condition is not so likely to be mistaken for the glomerular as for the tubular form of nephritis. Its differential diagnosis has already been discussed. Its essential features are the cyclic character of the albuminuria, and the absence of any other signs of disturbance of the renal function.

Retention of Urine.—This condition can easily be distinguished from the anuria of glomerulonephritis, by percussion of the bladder and by catheterization.

Primary Anemias.—In these diseases a condition of general anasarca is often seen, which closely resembles the edema of glomerulonephritis. The edema is probably etiologically as well as clinically much like the toxic edema described above as occurring in infants. The recognition of the primary disease is the most important guide in diagnosis. Furthermore there is no marked diminution in the excretion of urine, and slight if any signs in the urine of an inflammatory reaction in the kidneys.

Chronic Interstitial Nephritis.—True chronic interstitial nephritis, which is so common in adults, and in which the primary lesion is an arteriosclerosis of the vessels of the renal cortex, is extremely rare in childhood. Nevertheless I frequently find diagnosed as chronic interstitial nephritis, cases which are really chronic glomerulonephritis. The mistake comes from basing the diagnosis of the various diseases of the kidney too exclusively on the urinary findings. In the inactive stage of chronic glomerulonephritis there is little or no diminution in the quantity of urine, no edema, few renal elements in the sediment, and the quantity of albumin may be quite small. This urinary finding is much like that of the vascular nephritis of adults. The essential point in diagnosis is the existence of a previous attack characterized by edema. Whenever there is a history of a previous acute nephritis complicating an acute infection, or whenever there is a history of renal edema at any previous time, the diagnosis should be chronic glomerulonephritis, not chronic interstitial nephritis. Such a diagnosis does not imply that there are no extensive fibroid changes in the interstitial tissue of the kidney. Such changes may be present in chronic glomerulonephritis, as described under pathological anatomy. Only, the interstitial lesions are not due to primary vascular lesions, but are secondary to a toxic inflammatory process.

Unfortunately a definite history of previous edema or a previous acute attack cannot always be obtained. In such cases the diagnosis is doubtful. The previous attack may have been so mild that the edema was overlooked and the urine not examined. True primary chronic interstitial nephritis is a possibility, but chronic glomerulonephritis is always more probable in childhood.

It is often necessary, for purposes of prognosis and treatment, to differentiate between *acute* and *chronic* glomerulonephritis. Whenever the nephritis develops as a complication of an acute infectious disease in a child previously well, with a dark, smoky, or bloody urine, the nephritis may be considered acute. Whenever the nephritis develops insidiously, without an immediately antecedent acute infection, and without blood in the urine, the nephritis may be considered chronic, even though no history can be obtained of a previous acute attack. If such a history can be obtained, the diagnosis of chronic glomerulonephritis is all the more certain. The principal difficulty lies in distinguishing an acute exacerbation of a chronic nephritis from an acute nephritis. In both conditions the examination of the urine is about the same, revealing the presence of a considerable quantity of blood. That the attack represents an acute exacerbation of a previously existing chronic process can only be recognized from the previous history of the case. If there is a history of a previous acute attack, or if any previous routine examination of the urine has revealed any evidences of nephritis, it is probable that the condition is an acute exacerbation of a chronic glomerulonephritis. If no such evidence of a previous process exists, the physician is justified in regarding the case as acute glomerulonephritis.

PROGNOSIS. ACUTE GLOMERULONEPHRITIS.—In the acute form of glomerulonephritis, the prognosis has two aspects, the first being the outlook as to danger to life, the second being the outlook as to complete and permanent recovery.

The prognosis as to danger to life depends upon several factors, among them being the age of the child, the nature and severity of the primary infection, and the character and extent of the lesions in the kidney. The great majority of the typical cases occurring in children older than two years, recover from the acute attack. In most of these cases the principal clinical feature is edema, which is very rarely marked enough to cause edema of the lungs, or serious embarrassment to the circulation. Even in severe cases with uremic symptoms, recovery is the rule in childhood, although the prognosis must be more guarded. Occasionally, in a severe case complicating scarlet fever, death occurs from uremic poisoning. In infants, the prognosis is also favorable when the nephritis appears in a typical clinical form as a sequela of a definitely recognizable infection. When death occurs in such cases, it is usually due to feeble vitality or to some constitutional weakness from other causes. In the atypical, apparently primary cases in infants described above, the outlook is more unfavorable. The majority of cases in infants with marked constitutional disturbance, die.

What is the outlook as to the subsequent development of chronic glomerulonephritis in children who recover from the acute form of the disease? I think it would be a fair statement that the chances of a complete recovery on the one hand, and of the development of chronic nephritis on the other, are about even. During and after convalescence, as long as any albumin or renal elements are found in the urine, the prognosis as to permanent recovery must be very doubtful. Albumin and renal elements may persist for months or even years, and then finally disappear permanently. On the other hand most cases in which the urine remains abnormal for a long period eventually develop the clinical manifestations of chronic glomerulonephritis. If albumin and renal elements disappear quickly during convalescence, permanent recovery is highly probable, though not absolutely certain. If they disappear more slowly, permanent recovery is probable, but a favorable prognosis is not so certain.

The uncertainty of the prognosis is due to the fact that whether or not chronic nephritis will develop depends on the extent of the damage to the renal parenchyma. When the injury is great, the fibroid changes and glomerular sclerosis which cause the symptoms of chronic nephritis are more likely to occur. There is no certain clinical means of estimating the extent of this damage. It is very possible that the newer laboratory methods of estimating renal function may be of value as a guide to the prognosis in cases convalescing from an attack of acute glomerulonephritis. The most available test for general renal function appears to be the phenolsulphonephthalein test described in Volume I, page 143. This test has not yet been sufficiently tried with children to give a basis for conclusions as to its value in the acute nephritis of early life. It is, however, highly probable that its value in prognosis will be proved.

CHRONIC GLOMERULONEPHRITIS.—The prognosis of chronic glomerulonephritis as to complete recovery is unfavorable. There is, however, no definite duration of the disease. The disease may remain in the inactive stage for years, but sooner or later the symptoms almost invariably return. It is difficult in any stage of the disease to form a judgment as to the probable duration of life. The physician can only say that complete recovery rarely occurs, and that although the patient may live free from symptoms for years, an unfavorable ending is likely to occur, and this may happen at any time.

In the active stage of the disease, edema with marked diminution in the amount of urine may persist for weeks or even months, and then improvement may occur. As long as there are no marked symptoms of uremic poisoning, the outlook for such eventual improvement is fairly good. An increase in the quantity of urine and a diminution of edema are favorable signs. A fall in the amount of urine and an increase of edema are unfavorable signs. Even when improvement occurs, and the patient enters the inactive stage, a recurrence of active symptoms may occur at any time. On the other hand, when during the active stage uremic symptoms develop in addition to edema, the immediate outlook becomes more unfavorable. The uremic attack may be recovered from, or may prove fatal.

In the inactive stage of the disease, it is very difficult to predict the duration of freedom from active symptoms. The passage of a large amount of urine of low specific gravity is a bad sign in this stage, indicating extensive fibroid changes. The quantitative examination of the urine for urea is probably of some value in estimating renal function in this stage. It is also probable that the phenolsulphonephthalein test is of value in prognosis in this stage of the disease, although this has not been finally proved in children.

The liability of children with chronic nephritis to severe intercurrent infections, must be taken into consideration in estimating the prognosis.

TREATMENT.—While there are many points in common in the treatment of acute and chronic glomerulonephritis, it is more convenient to consider them separately.

TREATMENT OF ACUTE GLOMERULONEPHRITIS

PROPHYLAXIS.—Prophylaxis is important in acute nephritis, particularly in connection with the form which follows scarlet fever. The essential feature in prophylaxis is the reduction of the relative amount of toxins circulating in the blood. This is accomplished by favoring elimination through the skin and bowels, and by giving enough fluid to keep the toxins in the circulation well diluted. Patients having an acute infectious disease should be bathed daily as a routine, and if signs of renal irritation appear in the urine, should receive additional warm baths or spongings. The bowels should be kept freely open, and in scarlet fever sufficient saline cathartic should be given to cause one or two loose movements daily. The diet in the acute infections consists mainly of milk and farinaceous foods. If milk is refused, or is not well borne, the physician should see to it that some other liquid food is substituted, such as whey, buttermilk, or thin gruel mixed with milk. Plenty of water should be given throughout the course of an acute febrile disease. In scarlet fever these measures should be continued until five weeks have elapsed since the onset; in the other acute infections they should be continued until convalescence is fully established.

FLUID INTAKE AND DIET.—When the condition of acute glomerulonephritis has developed, there is no treatment which specifically influences the lesions in the kidney. Treatment is directed at the disturbance of renal function caused by the lesions. In the literature of the treatment of acute nephritis, two opposing methods will be found advocated. Some authorities advocate the forcing of fluids, while others advocate the restriction of fluid intake. The conflict arises from the fact that the disturbance of function in acute nephritis has more than one aspect. There is an accumulation of the toxic products of metabolism in the blood, and the giving of a large amount of fluid is intended to dilute these toxins and prevent the occurrence of uremic poisoning. The other aspect is the deficient power of the kidney to eliminate water and salts, and the restriction of fluid intake is intended to diminish the accumulation of fluid in the tissues and serous cavities. How shall we decide between these two conditions?

In children as distinguished from adults, acute nephritis is much more likely to be characterized by edema than by uremic symptoms. Therefore in average cases without uremic symptoms, the forcing of fluids is not indicated. Even with mild symptoms of uremic poisoning, such as vomiting, headache, and apathy or nervous irritability, the giving of additional fluid should only be considered if the symptoms persist after the efforts to increase elimination through the skin and bowels have failed. But in average cases with edema as the chief symptom, should the fluid be restricted? It is hard to answer this question. In general, I think that in medicine there is a tendency to go too far in applying the partial facts which we have learned about complicated disturbances of function to practical treatment. Our knowledge being only partial, the unknown features may cause any radical method of treatment to do more harm than good. In many cases of functional disorder I believe it to be safer to place a child under the hygienic and dietetic conditions of a normal routine, and not to adopt a plan based on partial facts or exclusive theories. But do we know enough about the functional disturbance in nephritis to proceed more boldly?

I think that a moderate restriction of fluid intake is advisable in acute nephritis characterized by edema without uremic symptoms. This is accomplished as a routine by cutting out water from the diet, giving the necessary amount of fluid in the form of milk. The indications for giving more than this amount of fluid will be considered below. The quantity of milk to be given depends upon other considerations, such as the age of the child, and its caloric and protein requirements.

In arranging a diet which will be best suited to the functional disability of the kidneys, or at least to what we know of this functional disability, we must remember that it is the products of *protein* metabolism which are excreted with most difficulty by the damaged kidneys, and which may be toxic if sufficiently accumulated in the blood. On the other hand, a certain amount of protein in the diet is required to balance the necessary nitrogenous metabolism of the body, and this amount, known as the minimum protein requirement, is about 1.5 grammes of protein per kilogram of body weight. This quantity should be given, but no more. The required protein should all be given in the form of milk, no meat, fish, or eggs being allowed. This quantity of milk will contain the moderately restricted fluid indicated as a routine.

To determine how much milk should be given in twenty-four hours, the following procedure should be employed:

a. Reduce the weight of the child from pounds to kilograms. This can be done by multiplying the weight in pounds by 440 and dividing by 1000.

b. Multiply the weight in kilograms by 1.5. This gives the grammes of protein to be given daily, to meet the minimum protein requirement.

c. Divide this minimum protein requirement by the amount of protein in 1 c.c. of milk. This amount of protein is found by dividing the percentage of protein in the milk by 100. If whole milk can be given (as it can in all cases except in infants requiring modified milk), the minimum protein requirement is divided by .035. In infants on modified milk containing 1.5 per cent., 2 per cent., or

2.5 per cent. of protein, the minimum protein requirement is divided by .015, .02, or .025. This will give the twenty-four hour quantity of milk required.

For example, suppose it is desired to arrange a diet in acute nephritis for a child of three years weighing thirty-two pounds:

(a) 32 × 440 = 14080 grammes, ÷ 1000 = 14 kilograms.
(b) 14 × 1.5 = 21 grammes, minimum daily protein requirement.
(c) 21 ÷ .035 = 600 c.c. or 20 ounces of milk daily.

The diet must not consist exclusively of milk. The quantity of milk which fulfils the minimum protein requirement, and meets the indication as to restricted fluid intake in cases with edema as the chief symptom, does not fulfil the caloric requirements of the child. A child of three years, weighing thirty pounds, cannot be properly nourished on 20 ounces of milk a day. This quantity of milk contains only about 420 calories, and the minimum caloric requirement for this child in health would be about 1200 calories. (See Vol. I, page 280.) In acute nephritis, or indeed during any acute illness, it is not necessary that the child take the full minimum caloric requirement, but it should not take very much less. This child should have 900 or 1000 calories in any event. The calories not provided by the milk are obtained from foods containing fats, sugars, and starches. The foods containing chiefly these food elements which are normal in the diet of a child of that age should all be given, so that practically the only dietary restriction in older children is the forbidding of meat, fish, and eggs. The green vegetables which contain comparatively little starch and sugar should also be restricted, in order that sufficient calories be taken without overloading the stomach.

The exact arrangement of the diet depends on the age and digestive powers of the child. Older children may have bread, butter, olive oil, cereals, potato, and any starchy vegetables they can digest. In infants the diet is necessarily more limited. It sometimes happens that with restriction of milk to the demands of the minimum protein requirement, the child cannot digest enough of the carbohydrate foods to meet the caloric requirement. In such cases, provided that there is no difficulty in digesting cow's milk fat, cream may be substituted for part of the milk. This increases the caloric intake without increasing the protein and fluid intake.

I can only indicate the general principles which govern the dietetic management of a case of acute glomerulonephritis. The details vary with each individual case. The general principles may be summarized as follows, for average cases with edema, but without uremic symptoms:—

1. Eliminate water from the diet.

2. Give enough milk to cover the protein requirement of the individual child.
3. Give no other protein foods, such as meat, fish, or eggs.
4. In addition to milk, give enough of the foods containing fat and carbohydrate to make up the caloric requirement in acute illness, arranging the diet in accordance with the age and digestive powers of the child.

In cases with toxic symptoms, there should be no restriction of fluid intake as a routine. In these cases the diet should be arranged as planned above, enough milk being given to cover the protein requirement, while the caloric needs are met by giving the articles of diet containing fats, starches and sugars which are suitable for a child of that age, or by substituting cream for part of the milk. The amount of milk needed to fulfil the protein requirement contains less water than the child would take under ordinary circumstances, and if no other fluid be taken, it means a restriction of fluid. This can be avoided by giving a certain amount of thin gruel, and by allowing the child to take as much water in twenty-four hours as it would take under ordinary conditions. This should be sufficient to dilute the poisons in the blood. I do not believe in forcing fluids as a routine when only mild uremic symptoms are present. This measure should only be used when serious uremia is threatened, and its indications will be considered below. The dietetic treatment of cases with mild uremic symptoms differs from that of the cases characterized chiefly by edema, only in that the giving of a normal amount of fluid is allowed.

HYGIENIC TREATMENT.—In cases of average severity, no special therapeutic measures are indicated. The child should be kept in bed, under the general conditions of hygiene and nursing suitable to the treatment of an acute disease. Care should be taken that no urine be lost, for the careful measuring and recording of the quantity of urine passed each day is very important in guiding the treatment. A careful record should be kept of all water and food given, so that the physician will know at all times just what is the fluid intake, the protein intake, and the caloric intake. A daily warm bath should be given, and at other times, sponging with warm water followed by the wrapping of the child in a blanket. Flannel night clothing should be worn.

The bowels should be kept freely open, preferably by the use of a saline cathartic. The best preparation in nephritis with edema is magnesium sulphate in doses sufficient to produce from one to three loose movements daily. If in average routine cases magnesium sulphate produces more purging than this, some milder saline, such as Carlsbad salts or Sprudel salts, should be substituted.

Under this treatment, most cases of acute nephritis will recover with due rapidity, and without showing alarming symptoms. *Diuretics are strictly contraindicated.* Iron may be given to combat the anemia, provided that it does not upset the stomach. More strenuous therapeutic measures than those outlined above are only indicated under special conditions.

TREATMENT OF SEVERE CASES.—More vigorous treatment is required in severe cases with very scanty urine, and either marked dropsy or general constitutional disturbance. The most important thing in such cases is to maintain free diaphoresis. This is accomplished by means of *hot packs*, given as described in Vol. I, page 155. Counterirritation over the region of the kidneys, obtained by means of the *mustard paste*, is useful. The bowels should be kept freely open, but harm may be done by too vigorous purging. The amount of cathartic given should be sufficient to produce three or four loose movements daily, but no more, and the effect of the purging on the general condition should be carefully watched. Epsom salts is the best cathartic in this condition. If Epsom salts cannot be taken without causing marked resistance on the part of the patient, or if vomiting is produced, compound jalap powder, or compound liquorice powder may be substituted, in doses proportioned to the age of the child. Calomel is contraindicated in nephritis. For a child of three years the dose of compound jalap powder is fifteen grains, and of compound liquorice powder one to two teaspoonfuls.

This more active treatment is continued until the quantity of urine secreted is showing a steady increase, when the routine treatment is resumed.

SUPPRESSION AND UREMIA.—Whenever there is total suppression of urine, the most effective therapeutic measure is the giving of rectal injections of normal saline solution. Two quarts, at a temperature of 105° F., should be injected high into the colon, and the injections should be repeated every six hours until the urinary secretion is reëstablished. All the measures indicated in the treatment of severe cases should be continued. The rectal injections should be begun with the advent of suppression, if possible before actual uremic symptoms have developed.

When actual uremia, as shown by marked apathy, stupor, delirium, convulsions, vomiting, diarrhea, and a pulse of high tension, comes on, the most effective additional therapeutic measure is venesection, followed by the intravenous injection of normal salt solution. The amount of blood withdrawn depends on the age of the child, his general condition, and the urgency of the symptoms. In a child of three years the amount should be from three to six ounces. The quantity of normal salt solution given intravenously should be twice as great as that of the blood withdrawn. In all uremic conditions, all restrictions of fluid intake should be omitted, and the child should be made to take as much fluid as possible. The nervous manifestations of uremia are best relieved by sodium bromide and chloral, given by rectum, in doses proportioned to the age of the patient.

COMPLICATIONS.—The complications which require special treatment are dropsy of the serous cavities, and edema of the lungs. If there is a marked ascites, with sufficient accumulation of fluid to distend the abdomen and push the intestines into the upper half when the patient is in a half-sitting posture (see Fig. 241), paracentesis should be performed and the fluid removed. Hydrothorax should be treated in the same way. If edema of the lungs develops, atropin should be given subcutaneously in doses proportioned to the age of the child. The most effective measure in pulmonary edema with a pulse of high tension, is venesection.

CONVALESCENCE.—The management of convalescence is important for the prophylaxis of chronic nephritis. The diet should be carefully restricted for a long time, certainly as long as any albumin and casts remain in the urine, and preferably for at least a year longer. Nitrogenous food must be restricted. It is not necessary to continue to give no more than the minimum protein requirement, and the child may take as much milk as he likes. The protein, however, should consist in the amount contained in milk and the small amount contained in the ordinary farinaceous foods and vegetables. Meat, eggs, and fish should not be allowed.

Special precautions should be taken against exposure to cold or to sudden changes of temperature. Flannel should be worn next to the skin. Every measure tending to promote good general nutrition should be utilized. Iron is required to combat the anemia. If with the approach of winter, the urine is not entirely normal, the child should, if possible, be sent to pass the winter in a warm climate. This is advisable in the winter succeeding an attack of acute nephritis, even if recovery has been apparently complete.

TREATMENT OF CHRONIC GLOMERULONEPHRITIS

THE ACTIVE STAGE.—The active stage of chronic glomerulonephritis is characterized by the same symptoms of renal disability as are seen in acute glomerulonephritis, namely diminution in the urinary secretion, edema, and the occasional development of uremic symptoms. The treatment is much the same as that of the acute form of the disease. In cases characterized only by edema, the same routine as to diet and restriction of fluid intake should be adopted as in acute nephritis; the hygienic routine, including rest in bed, careful recording of the urinary output and fluid and food intake, warm baths and spongings, and the promotion of free watery evacuation of the bowels, is the same as in the acute cases. The edema in chronic glomerulonephritis is often very persistent. In acute nephritis the symptoms are caused by the accumulation of the products of inflammation, which are soon absorbed with relief of symptoms. In chronic nephritis the symptoms are caused by permanent destructive changes in the renal tissues, and consequently the symptoms of functional disturbance tend to persist much longer. The only way of getting rid of the dropsy is by the persistent promotion of free diaphoresis and of a moderate watery catharsis. Hot packs, which are effective in obtaining diaphoresis in acute nephritis, often fail in chronic nephritis, particularly after continued use. It is often difficult to produce free sweating. In such cases, *hot air baths* should be substituted for hot packs. The frequency and duration of these baths must depend on the reaction and general condition of the individual patient. The indication is to produce a maximum of diaphoresis with a minimum of circulatory depression. Some children do not stand hot air baths well, and in such cases hot packs must be used.

It must be remembered that *the giving of pilocarpine is most strictly contraindicated,* in spite of the fact that it is the only really effective diaphoretic drug. The danger from edema of the lungs more than counterbalances any possible benefit from the use of pilocarpine.

It must also be remembered that *diuretics,* such as digitalis, caffein, calomel, theobromin-sodium salicylate, theocin, and potassium acetate, *are contraindicated* in the treatment of edema from nephritis. In acute nephritis diuretics are ineffective, because the glomeruli are blocked by inflammatory products. In chronic nephritis stimulation of the undestroyed portion of the renal parenchyma by diuretics is possible, and the giving of a diuretic may produce a marked increase in the quantity of urine secreted. Some authorities believe that diuretics can be advantageously used in chronic cases. Nevertheless recent research in the experimental nephritis of animals seems to show that the lives of such animals are *shortened by the use of diuretics,* and in my opinion it is better to avoid their use. In dealing with the persistent edema and scanty urinary secretion of the active stage of chronic glomerulonephritis, we must depend upon moderate restriction of fluid intake combined with the activity of the skin and bowels. In chronic as in acute nephritis, three or four daily watery evacuations should be obtained, but excessive purging should be avoided.

With the development of uremic symptoms, the restriction of fluid intake should be omitted. The development of an actual uremic condition is treated in the same way as in acute nephritis, with high rectal injections of normal saline solution, the forcing of fluids, hot packs or hot air baths, free but not excessive catharsis, and in severe cases, venesection with intravenous injection of normal saline solution. Excessive or persistent ascites, and hydrothorax, are treated by paracentesis. Edema of the lungs is treated as in acute nephritis, with atropin and venesection.

THE QUIESCENT STAGE.—In this stage of the disease the patient shows evidences of nephritis in the urine, but is free from edema or uremic symptoms. The general treatment is that which was described in connection with the convalescence from acute nephritis. Exposure, both to cold and to the contagious infections, is especially to be avoided. An active life in the open air is desirable. These indications can best be met if the patient passes the winter in a warm dry climate. All the details of hygiene tending toward good nutrition and robust development, should be attended to. Exercise should be regular, but should not be carried to the point of fatigue. There should be during the day, periods of rest in a recumbent position. Massage is useful in undeveloped children. Iron should be given for anemia, and bitter tonics, such as nux vomica, if the appetite is poor. The diet should not contain excessive salt, and meat, fish and eggs should not be given, if plenty of milk can be taken.

Occasionally there is seen in childhood a type of chronic glomerulonephritis in which the clinical manifestations are much like those of chronic interstitial nephritis in adults. A rare case of this type may represent a primary chronic interstitial nephritis, but the majority are cases in which the primary lesion was glomerular, but in which fibrous interstitial changes have reached an advanced stage. This is the *secondary contracted kidney* of the German writers. The clinical manifestations of importance are the passage of a large quantity of urine with very low specific gravity, the absence of edema, the presence of high blood pressure and a pulse of high tension, and the occasional occurrence of secondary cardiac hypertrophy, or of cardiac dilatation and insufficiency.

In these cases, in addition to the routine treatment of chronic glomerulonephritis, the condition of the circulatory system must be considered. Nitroglycerin may be given to reduce an excessively high blood pressure, but it must be remembered that children bear the nitrites proportionately much less well than adults, and the giving of nitroglycerin should be begun very cautiously in very small doses, controlled by frequent observations of the effect upon the pulse and blood pressure. The treatment of cardiac insufficiency is the usual treatment with cardiac stimulants, which has been described under the diseases of the heart.

Surgical interference has been recommended for children with chronic glomerulonephritis. The operation consists in decapsulation of the kidneys. It should only be considered in cases growing progressively worse under medical treatment. There is considerable immediate operative risk. Some writers have reported cases which appeared to have been permanently cured by this operation. In most of the reported cases in which the patient survived the immediate operation, the cure was not permanent, most of the children dying within a year after the operation. Striking temporary benefit, however, was seen in many cases. In a disease with so unfavorable a prognosis as chronic nephritis in children, any procedure of holding out any hope of benefit should be at least suggested to the child's parents.

PROBLEMS AND RESEARCH.—The last decade has seen great activity in experimental research in the subject of nephritis. The practical results of this work have been applied to the study of nephritis as it occurs in adults rather than in children, and we have seen that the nephritides of adults include an important pathological factor which is not found in early life. The value in the nephritis of early life of recent studies on renal function, remains to be determined. It is important, however, that the pediatrist should have a general idea of the progress which has been made, and of the problems awaiting final solution.

EXPERIMENTAL NEPHRITIS.—The foundation of modern research in nephritis is the production of experimental nephritis in animals. It has been known that the injection of various chemical poisons into animals will produce degenerative lesions of the renal epithelium. Among these substances are the chromium salts, the uranium salts, the tartrates and mercuric chlorid. On the other hand certain other poisons produce anatomical changes chiefly in the vessels of the glomerular tufts; among these substances are cantharidin, arsenic, and snake venom. From the anatomical standpoint a division has been made into epithelial and vascular renal poisons, but this distinction has been proved to be in no way absolute.

The production of experimental nephritis, with the discovery of a certain degree of correspondence between anatomical lesions and particular poisons, has stimulated research in various directions. In the first place, there is a possibility that the continued collecting and cataloguing of the various exogenous substances which can produce kidney lesions may throw some light on the nature of the bacterial and endogenous poisons which are the recognized cause of nephritis. In the second place, while albuminuria is the only constant symptom of nephritis, the other factors of kidney function have been found to vary in every direction from that of hyperactivity on the one hand to that of extreme inefficiency on the other. These variations have greatly increased the confusion attending any attempt to classify and describe the nephritides. If through experimental nephritis it becomes possible to locate or reproduce any single factor at a time, there is hope that there may emerge from the confusion a certain amount of correspondence between the lesion and the disturbance of function which it produces. The establishment of any such correspondence would be of the highest value in guiding dietetic treatment. The production of experimental nephritis is of good promise not only in classification and description, but gives also a means of estimating the value of various clinical tests of renal function in diagnosis, prognosis and treatment. Finally, a means of estimating the value of diuretics is afforded by the experimental nephritis of animals.

FOREIGN PROTEINS AND THE KIDNEY.—The use of experimental nephritis in the study of the pathogenesis of human nephritis has been chiefly in experiments with foreign proteins and the rôle of sensitization in the production of nephritis. There is some evidence that the production of nephritis may depend upon a previous sensitization with a foreign protein.

CERTAIN ASPECTS OF RENAL FUNCTION RECENTLY STUDIED.—On the basis of tests of renal function in various forms of experimental nephritis, Schlayer distinguished two types, a tubular form in which there are no marked changes in the vascular reactions which control the excretion of water, and a vascular form in which the vascular reactions are first increased, later diminished, and finally abolished. While there is some evidence that in nephritis polyuria is due to increased vascular reactions, the theory that anuria is associated with absence of vascular reactions has not been confirmed.

Schlayer found that damage to the renal tubules apparently interfered with the excretion of sodium chlorid. More recent investigations suggest that sodium chlorid is excreted through the glomeruli by filtration, but that damage to the tubular epithelium permits its reabsorption: The excretion of water and sodium chlorid, which are both normal body constituents, is influenced by factors outside the kidney. For this reason Schlayer tested the excretion of other substances, not normal body constituents. He found that in experimental nephritis of the vascular type there is a lessened excretion of *lactose*, while in the tubular type, the excretion of *potassium iodid* is delayed. He advocated that the rate of excretion of these two substances be used clinically in human nephritis as tests of the functional damage to the vascular and tubular apparatus respectively.

Extensive scientific study has been devoted to the important renal function of the excretion of the nitrogenous waste products. As an excess of nitrogenous compounds in the blood is promptly eliminated by the kidneys, the excretion of these substances depends on the quantity of protein in the diet, and no conclusions as to renal function can be drawn from estimations of urea or total nitrogenous output in the urine. Even if both intake and output are determined, a very small difference if prolonged might lead to a marked accumulation of nitrogenous waste in the body. These facts have led to the perfection of chemical methods of estimating the quantity of non-protein nitrogen in the blood. The establishment of a normal standard gives us another method of measuring renal function in nephritis. The ability of the kidney to eliminate certain test dyes is the basis of the best known and most generally useful method of estimating renal function, namely, the phenolsulphonephthalein test. There appears to be a general parallelism between this test and the estimation of the non-protein nitrogen in the blood.

TESTS OF RENAL FUNCTION.—These studies of renal function have provided us with a number of available laboratory tests. The tests most commonly employed are the phenolsulphonephthalein test, the lactose test, the potassium iodid test, the sodium chlorid test, and the test for the retention of non-protein nitrogen in the blood. The technic of the phenolsulphonephthalein test has been described (Vol. I, page 143). In the lactose test, glycosuria having been excluded by the ordinary tests, a sterilized solution of 2 grammes of lactose in 20 c.c. of water is injected intravenously under the strictest antiseptic precautions. After four hours the patient is catheterized and the urine discarded. All urine is then collected and tested for sugar. If sugar be found after four hours, there is suspicion of lessened lactose excretion, and if after six hours, the test is positive, and the case may be classified functionally as vascular nephritis. In the iodid test, one-half gramme of potassium iodid is given by mouth. In health this entire amount should be excreted within forty hours; delay beyond this period is suspicious, and if the test in the urine for iodin remains positive beyond sixty hours, the test is positive, and the case is classified as tubular nephritis. The ordinary starch test for iodin may be used for examining the urine. The other tests require a specially equipped laboratory, and are too complicated for ordinary clinical use. For the details of all these tests, the reader is referred to the most recent text books on laboratory diagnosis.

VALUE OF TESTING RENAL FUNCTION.—The literature of the past few years and of the present time is full of reports of the value of these various tests of renal function. There have been as yet comparatively few autopsies upon patients whose renal function in diseased conditions has been studied by means of these tests. Even in experimental nephritis there are certain discrepancies between the results of functional testing and the lesions. In human nephritis the few attempts which have been made to correlate the chief anatomical lesions, and the chief functional deficiencies, have not been very successful. This is due to the fact that in human toxic nephritis the lesions are not confined so definitely to one part of the renal unit as in experimental nephritis; indeed, experimental uranium nephritis appears to be of a mixed type. Furthermore, it is very probable that marked functional changes can occur without demonstrable anatomical lesions.

Attempts have been made to classify the nephritides upon a basis of tests of renal function. Such a suggested classification divides the nephritides into three varieties: (1) Tubular, characterized by delayed potassium iodid and sodium chlorid excretion; (2) vascular, characterized by delayed lactose excretion; (3) azotemic, characterized by increase of the non-protein nitrogen in the blood. It does not seem to me at all wise to adopt such a basis for the classification of the nephritides. One great danger arising from the perfection of modern methods of physiological research, is that we shall get too far away from the sound foundation of pathological anatomy. There is a tendency on the part of the physiological chemists and those interested in functional pathology, to minimize the importance of tissue changes. Yet, until knowledge of functional pathology has advanced much farther than at present, the classification of disease processes must rest upon anatomical lesions as its primary basis.

The practical value of the laboratory tests of renal function remains at present a subject of discussion, and is awaiting further investigation, especially in children. It seems to be generally admitted that the phenolsulphonephthalein test is the best measure of general renal damage. It appears to give information of the functional power of the kidney as a whole, indicating the ability to excrete, not single substances, but toxins. It is of little value in diagnosis, as there is no relation between the test and the specific character of the anatomical lesions. There is evidence that it is of value in estimating the general damage done. It appears to be of most value in prognosis, but it has been used also to estimate the extent of renal involvement in cardio-renal cases, and it is considered to be a valuable indication of an approaching uremia. It must be remembered, however, that most of the evidence tending to establish the value of this test has been obtained in the nephritis of adults, particularly in the cardio-renal cases which are not seen in early life. Much remains to be done in the nephritis of children before even the prognostic value of the phenolsulphonephthalein test can be considered as certainly established.

Of the other tests, no definite conclusion as to practical value can yet be drawn. There is a prospect that further research may disclose some such value in the nephritis of early life. If so, the value of the tests will probably lie in guiding dietetic treatment according to the particular disturbance of function shown by the tests.

DIURETICS IN NEPHRITIS.—Another important result of the production of experimental nephritis in animals was the study of the effects of diuretics in nephritis, and the results of this study appear to be of real practical value. The recent literature of this subject is quite voluminous. The general evidence appears to be that the giving of various diuretics may produce a notable increase in the output of urine, chlorids and nitrogen. While various types of experimental nephritis react differently, there is no evidence of any permanent benefit from diuretics. There are, on the contrary, various evidences of harm, as shown in some cases by lowering of the output of phenolsulphonephthalein, and in other cases by shortening of the lives of the laboratory animals. Diuretics in nephritis appear to produce a condition of harmful renal fatigue.

UREMIA.—The true pathogenesis of the group of symptoms known as uremia has always been one of the great unsolved problems in medicine. With the utilization of the most modern methods of investigation, much attention is being devoted to this subject. The two principal theories as to uremic symptoms are, (1) that uremia results from retention in the body of normally formed toxic substances which should be eliminated in the urine, and (2) that uremia is due to abnormal toxic substances formed as a result of a metabolism perverted by the kidney disease. It seems certain that uremic symptoms are not all due to a single cause. Studies of the non-protein nitrogen in the blood have led to a separation into two general groups. In one group are cases in which the uremic symptoms are accompanied by a notable increase in the non-protein nitrogen in the blood. In the other group there is no such increase. In the first group are the cases with drowsiness, headache and vomiting. In the second group are the cases with convulsions. Nevertheless the exact nature of the toxic substances, and the true pathogenesis of the symptoms, remain unknown. This subject is being actively studied.

ACIDOSIS.—The occurrence of acidosis in nephritis has recently been recognized, and is a subject to which much experimental study is being devoted. The acidosis is not of the type described under that heading in Volume I, characterized by the formation of the acetone bodies, but is of the type shown by an increased titrable acidity of the blood and increased carbon dioxid content of the alveolar air, and characterized symptomatically by dyspnea. Its occurrence appears to show a general parallelism with the accumulation of waste nitrogen in the blood, and with the ability of the individual to eliminate phenolsulphonephthalein.

SALT RETENTION AND EDEMA.—The pathogenesis of renal edema has always been one of the much discussed problems connected with nephritis. This problem also has recently been attacked by means of the newer methods of physiological research. All the facts connected with the occurrence of edema in nephritis are as yet unknown. It is known, however, that edema is closely connected with salt retention. In normal individuals the excretion of sodium chlorid by the kidneys is sufficient to maintain a balance between the salt taken in with the diet and the salt output. In nephritis the ability of the kidneys to excrete sodium chlorid is affected to a variable but marked extent. Sodium chlorid under normal conditions may act as a diuretic, its excretion being accompanied by the excretion of a correspondingly greater amount of water. It has long been known that renal edema is accompanied by retention of salt as well as of water. It is probable that in some cases the retention of water is primary, and that of salt secondary, the latter being necessary in order to maintain a normal constitution of the body fluids. On the other hand, there is evidence that the sodium chlorid may play the primary rôle in such combined retentions. There is also evidence that in some cases sodium chlorid may be retained without retention of water. There is no doubt that from the practical standpoint the intimate relation between nephritic edema, the intake of sodium chlorid, and salt retention through impaired power of renal excretion, is of great importance.

The mechanism both of water and of sodium chlorid retention, is still too imperfectly understood. Furthermore, there is abundant evidence that salt retention and hydremia are not the only factors in the production of renal edema. There is evidence that there are important extra-renal factors.

The most popular theory has been that the accumulation of toxic products in the blood exercises an influence upon the blood vessels such as to increase their permeability. The only evidence in favor of this hypothesis is found in the fact that in experiments with certain vascular poisons the tendency toward edema has been increased. We cannot tell, however, whether these poisons act upon the vessels throughout the body, or have some action upon the renal vessels only. Experimental tests of vascular permeability in animals have as yet not been followed by uniform results. Much work will probably be done in the future along these lines.

It has also been suggested that nephritic edema may be due to a change in the chemical or physical character of the tissues themselves, of such a nature that they hold more water. The weight of the experimental evidence appears to be rather against this hypothesis.

I have endeavored in the brief summary given in the foregoing pages, not to give a complete resumé of recent research in nephritis, but rather to present a general sketch of the problems attracting most attention. My object is to present these problems in such a way that the physician who is keeping in touch with the literature which is chronicling the very active research now going on in the laboratories, will be able to understand the value and bearing of what he may read in the future.

V. INFECTIONS OF THE KIDNEYS

PYELITIS AND PYELONEPHRITIS

Pyelitis is an inflammation of the mucous membrane lining the pelvis of the kidney, usually due to infection. When the inflammation extends into the tubules which open into the pelvis of the kidney, the condition is called pyelonephritis.

ETIOLOGY.—Pyelitis may be primary or secondary.

PRIMARY PYELITIS.—The primary form of pyelitis is very much the more frequent, and is a very common disease in infants and young children. It is primary in the sense that there is no local pathological condition of the genito-urinary tract to which the inflammation is secondary. It may, however, occur in the course of any disease, being particularly common in the acute diarrheas. The etiological connection with these diseases is not very obvious, and pyelitis may occur entirely without evidences of disease elsewhere. It is almost exclusively a disease of infancy, but is sometimes seen in the earlier years of childhood. It is about nine times as common in girl infants as in boy infants.

The microörganism which causes this primary pyelitis of infancy is the *bacillus coli communis*. In the majority of instances the colon bacillus is the only organism found, but mixed infections are occasionally seen.

It has always been generally assumed that this form of pyelitis represents an ascending infection, the bacilli entering through the urethra and traveling up the urinary passages to the pelvis of the kidneys. This is a pure assumption, not based on any proof. The reasons why pyelitis has been regarded as of urinogenous origin, are the following: (1) The almost exclusive occurrence of the disease in infants whose soiled diapers provide a ready means by which colon bacilli can gain access to the urethra; (2) the notably greater frequency of the disease in female infants, in whom the short urethra provides an easier route of invasion; (3) the frequent association of the disease with acute diarrheal conditions, in which the large numbers of colon bacilli are constantly discharged into the diaper. On the other hand, there is evidence against the theory that pyelitis takes place by ascending infection through the urinary passages. It seems rather strange, under such a hypothesis, that the colon bacilli should skip the bladder, and should travel so far against the urinary current, to localize themselves in the pelvis of the kidney.

There is certain positive evidence which has recently been obtained, strongly suggesting that the bacilli in the intestine gain access to the blood or lymph circulation, and are thus brought to the pelvis of the kidney, where conditions are favorable to their development, or where they find a tissue for which they have a selective affinity. The pathogenesis of colon bacillus pyelitis must be considered an open question, which will be further discussed under Problems and Research.

SECONDARY PYELITIS.—This form of the disease is much less common than the primary, and may be seen at any period throughout childhood. It may occur as an associated condition with almost any local disease of the genito-urinary tract. It is regularly present with renal calculus, and may be found associated with congenital malformations of the kidneys or ureters, with renal new growths, with renal tuberculosis, and with perinephritic inflammations. It is sometimes seen as a complication of one of the acute specific infections.

In all these cases, a variety of microörganisms, including colon bacilli, streptococci, staphylococci, pneumococci, bacillus pyocyaneus, typhoid bacillus, and others, have been found in various combinations.

PATHOLOGICAL ANATOMY.—The common primary form is bilateral. When pyelitis develops secondarily to a local cause, the process is usually unilateral.

The process is primarily a catarrhal inflammation of the mucous membrane, with congestion, swelling, and exudation of serum and polymorphonuclear leucocytes. In many cases, the inflammation extends into the tubules, causing degeneration of the tubular epithelium. In protracted and severe cases, small abscesses may be found in the renal parenchyma. If there is any obstruction to the free outflow of urine there may be an accumulation of purulent fluid in the pelvis of the kidney, leading to the condition known as pyonephrosis.

SYMPTOMS.—In the common primary form, the only symptoms which occur with sufficient frequency to be considered characteristic, are *fever* and *pyuria*.

Fever is present in the great majority of cases, but occasionally the urine may show evidences of pyelitis without there being any fever. In typical mild cases there may be no more than a slight elevation of temperature of two or three degrees. In other less common cases the temperature may rise abruptly to 104° F. or even 105° F. The temperature curve is very variable, usually tending to present marked exacerbations and remissions, but showing nothing characteristic of the disease. The temperature chart may resemble that of any other acute infection, including even malaria. There are often periods of several days without fever in various stages of the disease, followed by fresh rises of temperature. In cases with high fever, a remarkable absence of apparent general constitutional disturbance in proportion to the fever, is often one of the notable characteristics of the disease.

The urine is usually turbid from the presence of pus. The amount of pus varies greatly, both in different cases, and from day to day in the same case. The amount of pus on settling may occupy from one to fifty per cent of the volume of the urine. There is usually enough pus present to be visible macroscopically, but occasionally cases are seen in which the only abnormality of the urine is the presence of a variable number of pus cells in the sediment under the microscope. The quantity of pus appears to bear no relation to the height of the fever, or to the severity of any other symptoms which may be present. Also the marked variations in the amount of pus which are seen from day to day bear no relation whatever to the variations in the fever seen on the temperature chart.

The quantity of urine secreted is usually slightly diminished. The reaction is usually acid. Albumin is present in proportion to the amount of pus, and to the severity of the tubular nephritis which may accompany the pyelitis. The sediment, when examined microscopically, contains pus cells, red blood corpuscles, and epithelium, and may also contain tube casts. The pus cells are the most numerous element in the sediment, are usually well preserved, and tend to occur in large clumps or masses. Red blood corpuscles are present only in acute cases, and in the primary form are not found in sufficient numbers to color the urine; in some of the secondary cases the urine may be slightly red or smoky. Epithelial cells are numerous, especially in the early stages of the disease. Round, conical, spindle-shaped, and long-tailed cells are found. Casts, when present, are usually short and of large calibre.

In these typical cases of the primary pyelitis of infancy, the general condition of the child is often surprisingly little affected in proportion to the height of the fever and evidences of inflammation found in the urine. Some loss of weight, that universal indication of disease in infants, usually occurs, but even this is usually slight in comparison with the apparent severity of the infection as shown by the temperature chart. Anemia is apt to develop in long-standing cases.

A certain number of cases are atypical, and show other symptoms besides fever and pyuria. There may be some disturbance of digestion, as shown by vomiting, diarrhea, or abnormal movements. There may be a more than usually marked general constitutional reaction accompanying the fever. *Chills*, occurring at irregular intervals, are occasionally seen, but are less common than in the secondary form of pyelitis. Nevertheless chills are very uncommon as a manifestation of disease in infants, and when they occur, should suggest pyelitis more than any other possibility in diagnosis. The respiration is sometimes notably accelerated in proportion to the temperature and pulse, suggesting the possibility of pneumonia. Nervous symptoms suggesting meningitis are sometimes seen, and pyelitis is one of the causes of "meningismus" in infants. With all the other symptoms of acute infection which may occasionally occur in primary colon bacillus pyelitis, there is a notable absence of any symptoms or physical signs of a nature to localize the infection in the genito-urinary tract.

The disease runs a course of very variable duration. Fever may last only a few days, or may persist for six or eight weeks. As remissions of several days may occur, a fall of temperature to the normal is no sign that the disease is over. Relapses and recurrences, even after longer afebrile periods, are often seen. After fever disappears, pus may still be found in the urine, microscopically at least, for a much longer period. During the febrile period the blood usually shows a leukocytosis varying from 13,000 to 30,000, and usually causes an agglutination of the colon bacillus even in high dilution.

Cases which occur in older children, which represent usually the *secondary form* of pyelitis, are more apt to show symptoms other than fever and pyuria. Chills occurring at irregular intervals, are often seen in these cases. In this form there are often localizing symptoms, particularly frequency of micturition, pain on micturition, and tenderness on deep palpation in the region of the kidney.

DIAGNOSIS.—In the past the pyelitis of infancy has been a disease very frequently overlooked by the general practitioner, on account of the comparative difficulty attending the obtaining from infants, particularly female infants, of a specimen of urine for examination. The comparative infrequency of primary nephritis in infants has produced a general impression that urinary examination is not of extreme importance at this period of life. Nevertheless the great frequency of pyelonephritis is the strongest argument for the necessity of urinary examinations in infants which could possibly be brought forward.

Pyelitis belongs in the class of acute infections which do not reveal any characteristic signs on routine physical examination. I have often been called to see in consultation cases in which the physician was at his wits ends to explain a continued febrile reaction in a child in whom the physical examination was always wholly negative. In such cases, if physical examination has included proper examination of the ears, pyelitis stands first as a possibility in infancy, particularly if the patient is a female. The diagnosis depends wholly on the examination of the urine. A perfectly clear urine in an acute case will usually exclude pyelitis as a cause of fever, but it is safer to make a careful examination of the sediment even if the urine be macroscopically clear. If the urine be cloudy, it is necessary to determine that the turbidity is due not to urates or phosphates, but to pus.

Owing to the lack of distinctive symptoms, pyelitis is not often mistaken for other infections. I have seen cases with chills mistaken for malaria, and dosed with quantities of quinin in an effort to reduce the temperature. Cases with rapid respiration have been mistaken for pneumonia, cases with meningeal symptoms have been mistaken for meningitis, and cases with prolonged fever in which neither the urine nor the white count was investigated, have been mistaken for typhoid fever.

PROGNOSIS.—The course of the disease is very variable. The prognosis as to life is very favorable. Death sometimes occurs in infants with pyelitis, but in such cases it is not often to be attributed to the infection of the kidneys, but to some other accompanying condition, usually some acute or chronic gastro-intestinal disturbance which has so profoundly affected nutrition that the occurrence of pyelitis is sufficient to turn the scale. Of sixty cases collected by Jeffreys, nine died, six of the urinary trouble. Langstein considers that 90 per cent recover. Personally I have seen only one case in which death appeared to be primarily due to the renal infection.

The rapidity of recovery is very variable. The statistics vary according to whether "cure" is defined as clinical freedom from symptoms, or cultural freedom of the urine from bacteria. Some writers believe that chronic nephritis may follow prolonged colon bacillus infection.

TREATMENT.—Three different methods of treatment will be found advocated in the literature of the pyelitis of infancy. These are:—

1. Making and keeping the urine alkaline.
2. The giving of hexamethylenamin, or some other urinary antiseptic.
3. Vaccine therapy.

The preponderance of opinion is in favor of the alkaline treatment. The theoretical foundation of this method of treatment is the fact that in alkaline media colon bacilli develop less actively, and phagocytosis by the leukocytes is more active. Alkalinity of the urine may be produced by the giving of potassium citrate, potassium acetate, or sodium bicarbonate. The quantity of potassium acetate or citrate sufficient to produce and maintain a marked alkalinity of the urine is likely to cause vomiting or diarrhea. I usually prefer to use sodium bicarbonate, in doses of twenty to thirty grains every four hours, according to the age of the patient. Potassium citrate or acetate may be given in the ordinary doses proportioned to the age of the child, and sodium bicarbonate may be given in addition in amount sufficient to produce the desired alkalinity of the urine.

It is an important part of the treatment to give plenty of fluid. In a comparison recently made at the Infants' Hospital between cases in one ward under the alkaline treatment, and cases in another ward under no other treatment than the forcing of fluid, the apparent clinical results in the latter were quite as good as in the former. Whether or not the alkaline or any other form of medical treatment is used, *an amount of fluid should be given sufficient to keep the specific gravity of the urine below 1012.* Even if the infant takes the amount of milk appropriate to its age, it will require plenty of water in addition, if the specific gravity of the urine is to be kept low. I usually order that as much water as the child can be got to take voluntarily be given between feedings. The specific gravity of each day's mixed urine is taken, and if at any time it rises above 1012, I order that water be forced by being given with the spoon. If the child does not take all its milk, it is usually necessary from the beginning to force fluid by means of the spoon.

Hexamethylenamin is the favorite urinary antiseptic in the pyelitis of infancy, although ammonium and sodium benzoate, calomel, sandalwood oil, creosote and salol have all been advocated by various writers. Hexamethylenamin should not be given in connection with the alkalies; its use in this way may be responsible for many of the unfavorable reports of its value. It has been shown that the antiseptic action of hexamethylenamin depends upon the liberation of formaldehyd in the urine, and that this takes place only when the urine is acid. It is best to give hexamethylenamin in connection with sodium benzoate or acid sodium phosphate, in order to insure sufficient acidity of the urine.

The reports in favor of hexamethylenamin in the pyelonephritis of early life are not very convincing. I should advise that the giving of alkalies and the forcing of fluid be tried first, and that hexamethylenamin be tried only in unduly resistant cases.

There are some reports in favor of the value in pyelitis of vaccine therapy, particularly when autogenous vaccines were used. The favorable result in these reports was mainly an apparent relief of symptoms. In a disease of such variable course, conclusions based on evidence of this kind cannot have very much value. The weight of evidence appears to be against the value of vaccine therapy. Vaccines do not appear to influence the results obtained by other methods of treatment in acute and subacute cases, to prevent exacerbations, nor to bring about recovery in chronic cases. Nevertheless, there is no harm in trying a stock colon bacillus vaccine or an autogenous vaccine in a resistant or chronic case.

The other measures of general treatment in pyelitis are those of any acute infectious disease of the same general severity.

PROBLEMS AND RESEARCH. CHANNEL OF INFECTION.—The principal subject of controversy in the pyelitis of early life is the channel of infection. Although, as stated under etiology, the weight of opinion favors the theory of an ascending infection through the urethra, bladder and ureters, I find that the pediatrists of the present day seem one after another to be swinging over toward the theory of a hematogenous channel of infection.

The arguments in favor of the urinogenous theory have been summarized under etiology. The original objections to this theory were the lack of any explanation for the usual absence of cystitis in these cases, and the long journey of the colon bacilli against the urinary current. Some writers have attempted to explain ascending infection on the theory that the bacilli are carried up to the kidneys through the lymphatics which follow the course of the ureters. This explanation cannot hold, because it is a known fact that the direction of the flow in these lymphatics is from the kidney downward.

According to the theory of *hematogenous* infection, colon bacilli enter the general circulation from the intestine, or possibly from the vagina, urethra, or bladder. These bacilli are excreted through the glomeruli of the kidney, but find a soil favorable to their development in the renal pelvis. Conditions which tend to increase the number of colon bacilli in the blood, or which tend to cause a concentration of the bacilli (that is, an increased number of bacilli in proportion to fluid) in the kidney, are the causes predisposing toward the development of pyelitis. This would explain the occurrence of pyelitis in connection with intestinal disease, by increased absorption of bacilli into the blood; it would also explain the occurrence of colon bacillus pyelitis as a complication of various acute infections, by increased concentration in the kidney. Another predisposing cause is possibly the injury exerted on the renal epithelium by toxins.

Considerable valuable evidence in favor of the hematogenous theory has recently been presented. It has been known that presumably healthy persons can absorb colon bacilli from the intestine and excrete them through the kidneys. The most striking work on this subject is that of Cabot and Crabtree. Their evidence consists mainly in the demonstration of the colon bacillus in the blood in the early stages of pyelitis or even before the appearance of symptoms, and also in the experimental demonstration of the excretion of colon bacilli through the kidney. It is true that their work was carried on mainly with the colon bacillus pyelitis of adults. Still, even in adults, pyelitis is more common in the female. Of great interest in this connection is a recent case in the Infants' Hospital. This baby had lobar pneumonia, and a blood culture was taken with the idea of obtaining the pneumococcus. The blood culture showed, not the pneumococcus, but the colon bacillus. Two days later a typical colon bacillus pyelitis developed.

The great difficulty which still confronts the advocates of the theory of hematogenous invasion is the admitted preponderance of cases in females. This has been explained on the ground of a greater tendency toward constipation in females, but this explanation certainly cannot hold for the pyelitis of infancy. Another explanation is that the vagina of the infant presents a mucous surface which affords a greatly increased opportunity for invasion by colon bacilli, and for their absorption into the blood.

There is also a theory of invasion from the intestine through the lymphatics, but this is not supported by any definite evidence.

TREATMENT.—The possibility of preventing pyelitis by means of prophylactic vaccination, is also receiving considerable attention. This is of more practical importance in adults than in children, because pyelitis in adults is more often secondary to surgical operations or to well-recognized local disease of the urinary tract.

A recent paper by Quinby on pyelitis in infants, contains an example of the sort of progress which is being made at the present day in the application of scientific methods to treatment. In some recent work by Clark, it was found that by testing the hydrogen ion concentration of the culture media on which the colon bacillus is grown, it is possible to divide the various strains of colon bacilli into two groups according to the degrees of acidity or of alkalinity compatible with their cultural life. Quinby suggests that this be used as a diagnostic test, and that according to its results we should endeavor to obtain in the urine a degree of acidity or alkalinity which lies outside the limits within which the particular strain of bacillus can develop.

SUPPURATIVE NEPHRITIS

(Abscess of the Kidney)

This is a condition characterized by the formation in the kidney of multiple miliary abscesses. It is rare in early life.

ETIOLOGY.—The microörganisms which are found in the lesions of this disease are those which are capable of producing suppurative lesions in any part of the body. Staphylococci have been found most often in children.

The usual route of invasion is through the blood stream. The bacteria circulating in the blood are caught in the renal capillaries, and in developing form miliary abscesses. The disease is probably always secondary to an infectious process in some other part of the body, although the primary focus of infection may not always be apparent. It may be part of a general pyemia, the renal lesions being due to the depositing in the kidneys of the infected emboli.

In rare cases, the lesions of suppurative nephritis have been produced by extension of an infectious process to the kidney by direct contiguity.

PATHOLOGICAL ANATOMY.—The process begins with the formation of multiple septic infarcts in the cortex of the kidneys. These soon break down under suppuration, forming small abscesses. Large abscesses may be formed by the confluence of smaller ones.

SYMPTOMS.—The principal symptoms are septic fever and pyuria. The temperature is irregular, as in septic processes of a similar character. Constitutional symptoms and prostration are variable, but are usually more marked than in colon bacillus pyelitis. Chills are not uncommon. In typical cases the urine contains much pus and many casts. There is often localized tenderness in the region of the kidney.

DIAGNOSIS.—Cases in children are usually overlooked, the condition being found at autopsy. The principal symptoms being fever and pyuria, they are usually attributed to the much more common pyelonephritis. Marked constitutional disturbance, marked prostration, and an appearance of general sepsis, should suggest the possibility of suppurative nephritis. Tenderness over the kidney is very rare in pyelitis, but is often both present and marked in suppurative nephritis. If cultures from the urine reveal one of the common pus-producing organisms, the diagnosis is greatly strengthened.

PROGNOSIS.—The disease runs a course of variable length, similar to other suppurative processes of the same nature. There is evidence that some cases have recovered spontaneously, with the formation of the lesions of secondary contracted kidney. Most cases die of sepsis, but on account of the difficulty attending certainty of diagnosis, an unqualifiedly unfavorable prognosis cannot be given.

TREATMENT.—Catheterization of the ureters should be employed if possible. In young children this is only possible in females. If it be found that the pus is coming from one kidney only, and that it contains pyogenic organisms other than the colon bacillus, surgical removal of the affected kidney is indicated.

If both kidneys are involved, the treatment can only be symptomatic and palliative. Hexamethylenamin together with sodium benzoate or acid sodium phosphate should be given. The general treatment is that of a severe septic infection.

PERINEPHRITIS

Perinephritis is an inflammation in the connective tissue about the kidney. It may end in resolution or suppuration. It has been confounded not infrequently with hip disease or vertebral caries. The most important paper on perinephritis in children was published in 1880 by Gibney, who reported twenty-eight cases.

ETIOLOGY AND PATHOLOGICAL ANATOMY.—Perinephritis in children may be secondary to suppuration in the kidney, such as results from calculi or tuberculosis, or to disease of the suprarenal capsules or to trauma. It is more frequently apparently primary. The cellular tissue about the kidney becomes inflammed, and in the majority of cases suppuration occurs. The pus may discharge outward in the ileo-costal space, or into the pleura or bowel.

SYMPTOMS.—The onset may be acute or chronic. In the acute cases there is high fever, often beginning with a chill. There is usually gastric disturbance, and the constitutional symptoms may be very severe. The bowels are constipated at times. There is usually local pain referred either to the hip or small of the back. The spine is painful and rigid, and the leg on the same side is drawn up as the movement of extension is productive of pain. General movements of the body are instinctively avoided by the patient. There is tenderness and resistance in the lumbar region, and later a tumor appears, in which deep fluctuation may sometimes be obtained. The size of the abscess may be very large. The cases which discharge into the pleura are usually diagnosticated as empyema. The character of the urine is rarely affected. The condition runs its course, in from a few weeks to several months.

DIAGNOSIS.—Hip disease is likely to be confused with perinephritis, but the diagnosis is easy after careful examination. In hip disease the process is much more chronic, and the deformity is produced insidiously. Moreover, the pain is lower down, and there is tenderness over the joints. There is limitation of all the movements of the hip instead of extension alone.

Perinephritis may give local signs similar to psoas abscess from spinal caries, but the characteristic changes and deformity of the lumbar vertebrae are absent.

Perinephritis may be confused with typhoid fever when the constitutional disturbance is severe and prolonged and the local signs develop slowly.

PROGNOSIS.—The primary cases almost always end in complete recovery if promptly recognized and treated. Peritonitis from rupture of the abscess is the most serious complication.

TREATMENT.—The treatment is surgical. Rest in bed and hot local applications are first indicated, and later incision with free drainage of the abscess.

TUBERCULOSIS OF THE KIDNEY

Tuberculosis of the kidney in children occurs in two forms: (1) As a miliary tuberculosis, the tubercle bacillus being conveyed into the tissues by the blood. This form rarely reaches any clinical importance. (2) As a tuberculous process beginning in the pelvis of the kidney and extending from this into the cortex. This form is by far the most serious in its symptoms and results. The detection of the tubercle bacillus in the urine in these cases is the only positive sign of the disease. The symptoms are those of a pyelonephritis represented by pus in the urine, an irritable bladder, and sometimes the presence of a tumor or abscess in the renal region. The treatment is essentially surgical. I have seen cases which apparently recovered without other treatment than that of careful feeding and hygiene and abundance of out-door life. Amyloid degeneration of the kidney may occur as a result of chronic tuberculosis, especially of the joints.

B. DISEASES OF THE BLADDER

I. CONGENITAL MALFORMATIONS

Malformations of the genito-urinary tract due to anomalies of embryonic development are not uncommon. As the development of all parts of the genito-urinary apparatus takes place from the same embryonic tissues, developmental anomalies are apt to involve more than one part, and malformations involving the bladder are usually combined with congenital lesions of the genitals. A great variety of rare anomalies have been reported. The malformation of the bladder having most clinical significance is that known as *exstrophy.*

EXSTROPHY OF THE BLADDER

In this condition there is in the anterior abdominal wall a median fissure or opening which is lined by the mucous membrane of the bladder. The fissure begins at the umbilicus, and in complete cases extends to the tip of the penis, including the anterior abdominal wall, the pelvic bones and the urethra. The pelvic bones may be entirely separated, or may be connected by a fibrous band behind the bladder. In some cases the fissure does not extend to the penis, but the external genitals are usually imperfectly developed. Exstrophy of the bladder usually occurs in males. An analogous condition is sometimes seen in females, in which the median fissure involves the clitoris and labia, the vagina usually being absent. In these cases the fissure may reach nearly to the anus, and the rectum may open into the prolapsed bladder.

In exstrophy of the bladder the hypogastric region is occupied by a red, slightly corrugated, velvet-like surface which represents all that exists of the mucous membrane of the bladder. It is kept constantly moist by the urine which trickles upon it from the openings of the ureters, which may be recognized in the lower lateral portions as slightly rounded elevations from which urine oozes. In complete cases this mucous membrane is continued into that lining a shallow furrow on the dorsal surface of the short penis.

These deformities are compatible with a long duration of life. Any exstrophy of the bladder, whether complete or partial, renders it impossible to prevent the clothing of the patient from being kept constantly soaked with urine. This gives rise to a strong ammoniacal odor which causes the patient to be a nuisance both to himself and to all about him. The skin surrounding the opening usually becomes excoriated.

Surgical operation for the relief of this condition should always be undertaken. The most effective operative procedure is transplantation of the ureters into some part of the large intestine, usually the rectum. The results of this operation are often remarkably good. The rectum becomes tolerant of the urine, retains it for hours, and evacuates it without discomfort. Pyelitis from ascending infection rarely occurs in these cases.

II. NEW GROWTHS

The bladder may be the seat of either primary or secondary new growths, but such cases are rare in early life. Most of the reported cases have occurred in children between the ages of one and five years. Sarcoma is the form of new growth most frequently seen. Most of the tumors start in the mucous membrane in the region between the openings of the ureters.

The symptoms of vesical tumor are hematuria, pain in the region of the bladder, and disturbance in the passage of urine.

The diagnosis is often difficult. Persistent hematuria may mean a new growth in either the kidney or the bladder. In females the diagnosis may be made with certainty by means of the cystoscope, but this cannot be used in very young males. Sometimes the tumor may be palpated through the rectum.

Tumor may be complicated by cystitis, pyelonephritis, hydronephrosis, or suppurative peritonitis. Even with operation, the prognosis is unfavorable.

III. FUNCTIONAL DISTURBANCES

ENURESIS

(Incontinence of Urine; Bed-wetting)

The term enuresis as used here refers to incontinence of urine occurring as a result of disturbance of the function of control of the bladder. Incontinence of urine occurring as a manifestation of some congenital malformation of the genito-urinary tract, such as exstrophy of the bladder, abnormal opening of the bladder into the vagina, persistence of the urachus, and so forth, is not included. Incontinence of urine is also seen as a manifestation of some organic lesions of the central nervous system. It is seen in idiocy, cerebral paralysis, malformations and injuries of the spinal cord, spina bifida occulta, meningitis, brain tumor, myelitis, vertebral caries, and similar conditions. Such cases will also not be included in the present consideration of enuresis.

The period at which the child normally learns control of the bladder, depends upon the training. In many cases it is possible to teach infants to control the bladder during waking hours before the end of the first year. More often this control is not acquired until some time in the second year. After voluntary control during the waking hours is acquired, children often still continue for a variable time to have nocturnal incontinence. Usually, however, the normal infant ceases to wet the bed at night, except perhaps at rare intervals, by the end of the third year.

The period at which the normal incontinence of infancy becomes abnormal cannot be arbitrarily fixed. For purposes of clinical description, we may consider as a case of enuresis a child who has incontinence by day during or after the third year, and nocturnal incontinence after the completion of the third year.

ETIOLOGY.—The literature of the etiology of enuresis of early life presents an almost hopeless maze of theories, each of which lays stress upon some particular aspect of the subject. It must be remembered that none of the various theories presented is supported by conclusive scientific evidence. A host of possible etiological factors have been deduced from the various conditions with which enuresis has been found associated. Such association is suggestive only of a possible etiological relationship, but is not proof. Again, the fact that enuresis ceases after the removal of some particular possible cause, is not conclusive proof that the enuresis was due to that cause; in a functional disturbance so closely connected with the nervous system as is enuresis, the influence of suggestion cannot be excluded whenever anything is done. Other evidence is drawn from the association of enuresis with certain constitutional tendencies in the patient or in his family. This form of evidence also is suggestive, but not conclusive.

Further confusion has been brought about by the use of an inexact terminology. It is often stated that enuresis is always a *symptom*, not a disease, or that in most cases it is a symptom, idiopathic cases being rare. If this means only that the incontinence of urine always has a cause, or usually has a cause, it is of course true. But enuresis is a real functional disturbance, having multiple causes similar to those of other functional disturbances which are always classified separately as diseases, and in this sense it should be considered a disease rather than a symptom. Only when enuresis appears as a common manifestation of some other separate recognizable disease entity, should it be considered as only a symptom.

The known facts as to the control of the bladder must necessarily form the basis of all theories as to the etiology of enuresis. The emptying of the bladder is accomplished mainly by the detrusor muscle in the wall of the organ, which is supplied chiefly by the fibres of the sympathetic nervous system. The voluntary control which resists the tendency of the bladder to empty itself is supplied mainly by the sphincter vesicae muscle, which is innervated by fibres from the third and fourth sacral nerves of the sacral plexus. The reflex arc is established by sensory nerves which enter the lumbar enlargement of the spinal cord in the usual manner, with the usual connections with the sympathetic ganglia. These nerve systems are in turn inhibited or stimulated by nerve fibres of the upper nervous system. The weight of evidence suggests that the infant acquires the power of resisting the tendency of the detrusor muscle to contract, not so much by a development of increased muscular power in the sphincter as by a development of the function of the nervous mechanism of control.

That the nervous mechanism may be affected by many external causes which send abnormal stimuli to the nervous system, is highly probable. It is not probable, however, that such abnormal stimuli are the sole cause of enuresis, because they are so very common that if they were the sole, or even the chief etiological factor, enuresis would be seen in most children, instead of in only comparatively few. We must assume that in enuresis there is some underlying etiological factor which is present only in the individuals who have the condition.

I believe that the fundamental underlying etiological factor in enuresis is *functional instability of the nervous system.* I am therefore most in agreement with those who believe that enuresis represents a specific neurosis. It is possible that relative weakness or loss of tone in the musculature of the vesical sphincter may play a part in certain cases, but the weight of evidence certainly suggests that the chief cause is to be found in a disturbance of the function of the nervous regulatory mechanism.

It is not enough to trace enuresis to a functional disturbance of the nervous system. We must go further, and consider the various causes which may produce the nervous instability. In the first place, there is evidence that there is an hereditary factor in many cases. The family history in cases of enuresis often gives abundant evidence of neuropathic tendencies, and the frequency of enuresis in "nervous" children is highly suggestive that a *neuropathic constitution* is an important etiological factor.

There is no doubt that there are many causes acting from outside the body, which can affect the nervous mechanism in such a way as to increase its instability and reflex excitability. The nervous system of the child as compared with that of the adult is normally unstable, and easily susceptible to functional disturbance produced by external factors. Among the external factors may be numbered all those *conditions of faulty hygiene and environment,* which are known to have so unfavorable an influence upon the development of the body as a whole. In such cases the delicate and susceptible nervous system is especially prone to suffer. The association of enuresis with nutritional disorders such as indigestion and rachitis, can be explained on this basis.

Another, and perhaps a more important cause of instability of the particular part of the nervous system which controls the bladder, is exaggerated sensory stimuli. The researches of Von Zeissl show the manner in which reflex causes may act in starting or checking the flow of the urine. Thus, a reflex carried to a proper center in the lumbar cord would, through the motor fibres of the erector nerve, contract the muscular coat of the bladder, and through the inhibitory fibres of the same nerve relax the sphincter vesicae. In this manner the urine which is being expelled by the contracting bladder is allowed to pass without hindrance through the relaxed sphincter vesicae. It is also to be remembered that the act of urination is in part under the control of the will. Admitting these anatomical and physiological facts, it is easily understood how the causes which produce enuresis may act in two ways: either directly on the centers in the lumbar cord, making them more irritable or unstable, and in that way increasing their reflex excitability, or indirectly through exaggerated reflex causes that affect both acceleratory and inhibitory influences sent to the bladder. These influences may be psychic, originating in the brain, or may be the result of external irritation originating in or near the bladder itself. There is also during childhood a lack of development of the centers of inhibitory reflex acts, and in this way the muscular fibres of the bladder, having no inhibitory restraint, are excited to action by even so slight a reflex cause as a small quantity of urine in the bladder. For this reason enuresis is a normal condition during infancy, and ceases when the child's inhibitory mechanism is more developed. The inhibitory influence of the will is in abeyance during deep slumber, and nocturnal incontinence is therefore more frequent than diurnal. In any diseases which are accompanied by anemia and malnutrition the reflex irritability of the lumbar nerve-centers is much increased, and enuresis may result.

The possible reflex causes originating in or near the bladder itself are numerous. *Excessive drinking*, by producing overdistention of the bladder, may be the cause. An abnormal irritability of the urine from *alkalinity*, or more often, from *excessive acidity*, is a possible cause. The cause of the abnormal urinary reaction is usually a faulty diet. Nevertheless recent researches have not found an association of enuresis with an abnormal chemistry of the urine as often as was formerly supposed to be the case. More common causes of reflex stimulation originating in the genito-urinary apparatus are such conditions as *phimosis*, *adherent prepuce*, *adherent clitoris*, and *balanitis*. Other possible causes are *vulvovaginitis*, *constricted meatus*, *masturbation*, *rectal parasites* (pin worms), *rectal polypi*, *fissure of the anus*, and *overloaded rectum*. There are also certain conditions recognized as distinct diseases, which can cause exaggerated stimulation of the genito-urinary tract, and in which enuresis appears as a recognized symptom. Among these conditions are *diabetes mellitus*, *diabetes insipidus*, *urethritis*, *cystitis*, *pyelitis*, *nephritis*, *vesical calculi*, and *vesical neoplasms*.

Among the possible reflex causes originating outside the immediate neighborhood of the genito-urinary tract, the two which have been most frequently mentioned are *hypertrophied tonsils or adenoids*, and *indigestion*. Both of these conditions are very common in early life, and I do not think that the rôle assigned to them in the etiology of enuresis is as important as is stated by many writers. In certain recent statistical investigations of enuresis, hypertrophied tonsils or adenoids could not often be definitely associated as etiological factors, and their operative removal did not cure the condition in many cases. They may be a cause, but probably not often. When operative removal brings relief, it is possible that the cure may be due to the suggestive influence of the surgical shock. As to indigestion, it is impossible to say whether this condition causes increased instability of the nervous system through reflex irritation, or whether it causes it through the effect of malnutrition upon the functional effectiveness of all parts of the body. I have found enuresis associated most often with the particular form of *chronic indigestion from* a relative excess of *carbohydrate*, which is seen in older children, and which is described in Division V.

Thyroid insufficiency has been recently suggested as a possible etiological factor. The ductless glands never escape being included as factors in conditions of unknown etiology. There is no conclusive proof of any connection between enuresis and the thyroid, the evidence being drawn from cases in which the condition improved under thyroid extract.

SYMPTOMS.—Enuresis may be nocturnal or diurnal, or both. The great majority of cases are either nocturnal only, or both nocturnal and diurnal. In ordinary enuresis the incontinence does not consist in a dribbling of urine, but in a complete emptying of the bladder. At night in some cases the child may wake up with an imperious desire to urinate, contraction and emptying of the bladder following immediately without the child being able to hold the urine back after feeling the impulse; in other cases a purely reflex contraction of the bladder may occur without wakening the child. In diurnal enuresis an irresistible contraction of the bladder begins as soon as the impulse to urinate is felt.

Incontinence may occur once every night, several times a night, or both every night and several times during the day. It may be only occasional, appearing under the influence of some special cause and continuing for a few days or weeks until the cause is removed. The condition may continue from infancy for an indefinite period, in many cases at least until the sixth or seventh year, and sometimes until puberty. True functional enuresis not due to malformation or to organic disease of the brain or cord, practically always ceases at puberty. Some cases are seen in which enuresis develops at a later period after control of the bladder has been gained.

PROGNOSIS.—In ordinary functional enuresis, the prognosis as to the duration of the condition is very variable. While it depends to some extent upon the age of the child, the duration of the condition, and the nature of the exciting cause, there are many unknown factors connected with the underlying condition of nervous instability, which render it impossible to give any definite prognosis in an individual case. In children coming under treatment at the age of from three to five years, a cure can be accomplished in many cases. The prognosis is also favorable in older children in whom the condition has not been present since infancy, but is of recent development. On the other hand, cases in older children in whom the condition has been present since infancy, are likely to be very resistant. When a definite source of reflex irritation, particularly a source in or near the genito-urinary tract, can be found and removed, the outlook is more favorable than when no such exciting cause can be found. There are a certain number of cases which obstinately resist all treatment until puberty, when the condition usually ceases spontaneously.

The prognosis of cases in which enuresis is only a symptom is that of the disease which causes it. In cases in which the condition is due to organic disease of the brain or cord, or to malformation of the genito-urinary tract, the outlook is hopeless unless the cause is amenable to surgical treatment.

TREATMENT.—The essential indication in the treatment of enuresis is the removal of all possible causes. The first step is a thorough physical examination. The physician should pay particular attention to the finding of sources of possible reflex irritation in or near the genito-urinary tract. The most important of these were enumerated under etiology. Adhesions of the prepuce or clitoris should be broken up, smegma should be removed, and the parts should be kept clean. Phimosis should be treated by circumcision. Even if without phimosis the prepuce be very long, circumcision should be performed, as it is often followed by improvement. Pinworms, and all other local conditions should receive appropriate treatment. No possible local cause should be forgotten in the clinical investigation of the case.

The urine should always be examined. This discloses such possible causes as pyelitis, cystitis, urethritis, and diabetes, as well as giving evidence as to whether the composition of the urine itself is a possible source of irritation. If the urine be concentrated and of high acidity, more fluid, especially water, should be taken, and one of the alkaline diuretics, potassium citrate or potassium acetate, should be given in doses proportioned to the age of the child. If the urine be alkaline, the diet should be regulated to contain a smaller proportion of farinaceous food, and sodium benzoate may be given. If the urine is passed in excessive quantity and is of low specific gravity, the quantity of fluid ingested should be restricted, especially toward bed-time. Indeed a restriction of fluid intake toward night, with a dry supper, is indicated in all cases of nocturnal enuresis. While these measures dependent on the urinary analysis should always be taken, the majority of cases will not be benefited by them.

The physician should also seek removable sources of reflex irritation outside the region of the genito-urinary tract. Nevertheless the removal of tonsils and adenoids has in my experience rarely produced a favorable effect, and I do not advocate the employment of this procedure for no other reason than that the child wets the bed.

While the removal of local sources of irritation is important, it has occupied so prominent a place in the literature of enuresis, that there is danger that the importance of certain general measures be forgotten. The underlying condition is the unstable nervous system, and this is often due to factors in hygiene and environment which are just as easily removed as are local sources of irritation. Every detail of the child's daily life should be carefully scrutinized, all faults should be corrected, and a regimen should be adopted in which such matters as diet, sleep, fresh air, and exercise are carefully regulated in accordance with the principles described in Division I. In the regulation of the diet it is not only essential to exclude all articles not part of the normal diet of a child of that age (see Division IV), and to forbid all eating between meals, but it is also advisable to exclude cane sugar almost wholly. Such conditions as anemia, indigestion, constipation, and malnutrition should receive careful attention.

The hygiene of the nervous system is particularly important. All overstimulation should be prevented, particular attention being paid to such excessive stimuli as overtaxing of a "teacher's pet" at school, the reading of exciting stories, and very violent or exciting forms of play. I have seen cases of enuresis cured by no other treatment than the careful attention to the details described under the Hygiene of the Nervous System in Division I. It is of particular importance in enuresis to *avoid using punishment*, whether corporal or not, *for the condition.* Good results, on the other hand, can sometimes be accomplished by rewards; "a nickel for every dry night" has cured some cases. With bright children it is advisable to talk with them about the condition in a matter-of-fact way, with a view to obtaining their intelligent coöperation. It is often possible to induce them to keep a record of wettings by certain pencil marks on paper, and they soon begin to take pride in reducing the number of marks.

In most of the cases which resist the removal of local causes, and which eventually also resist general hygienic measures, enuresis persists because it has become a habit. Habits can best be broken up by training. This is a most important part of the treatment of every case of enuresis. During the day the child should be made to void urine at regular intervals, these intervals being shorter than the average time between wettings. The intervals should be gradually lengthened, in order to accustom the bladder to hold larger quantities of urine. At night the child should be wakened regularly to void urine. If he wets more than once during the night, he should be wakened at first at three-hour intervals. Older children can often wake and pass urine themselves at the call of an alarm clock. Younger children must be wakened by a coöperating mother or nurse. The intervals are gradually lengthened, and often it is possible after a comparatively short time to obtain a dry night with one wakening at about 10 P. M. Children who wet only once should be wakened at some time previous to the usual time of wetting. Many cases can be cured by this method of training.

A certain number of cases will resist all the methods of treatment described above. The only additional therapeutic measure is the use of drugs. Drugs may be used at the start as an accessory to the other more important methods of treatment, or their use may be reserved for resistant cases. There is some evidence of a favorable effect from the use of drugs, but on the whole the results are likely to be disappointing. Too much dependence has been placed upon drugs in the treatment of enuresis, because it is so much easier to write a prescription than to carry out a painstaking investigation as to causes and a minute regulation of the child's habits of life. A host of drugs have been recommended for enuresis.

Belladonna has given the most evidence of effectiveness. It may be given either in the form of tincture of belladonna, or as atropin. The physician should begin with the doses scheduled in Division II as proper for the various ages, given three times a day, and should very gradually increase the size of the doses, watching carefully for dryness of the mouth or other toxic symptoms, which are a sign that the dose can not be further increased. I am accustomed to increase the dosage as shown in the table:

TABLE 74

Schedule for Administration of Belladonna in Enuresis in a Child of Five Years

DAYS	A. M.	M.	P. M.
1	♏ iii	♏ iii	♏ iii
2	♏ iii	♏ iii	♏ iv
3	♏ iii	♏ iv	♏ iv
4	♏ iv	♏ iv	♏ iv
5	♏ iv	♏ iv	♏ v
6	♏ iv	♏ v	♏ v
7	♏ v	♏ v	♏ v

And so on until dryness of the mouth or some other symptom develops

Strychnin is sometimes of benefit in cases of diurnal incontinence.

Thyroid extract has recently been widely recommended in the treatment of enuresis. There are some favorable reports, but the evidence is not very conclusive. The use of thyroid is based on the possibility of thyroid insufficiency being an etiological factor in enuresis, a theory which in my opinion is not very probable. I have had no personal experience with the use of thyroid extract.

PROBLEMS AND RESEARCH.—The literature of enuresis is very voluminous. For the most part it consists of theories which are supported by evidence of a wholly inconclusive nature. Each writer seizes upon some condition with which enuresis is found associated in statistical reports, and upon this basis builds up his theory of the etiology of the condition. In other instances evidence is presented of a statistical nature showing the percentage of improvement with various methods of treatment. All evidence of this kind is of comparatively little value. Unfortunately, enuresis is a condition which is not accessible to scientific experimental investigation. The only evidence available is statistical reports showing, for example, the percentage of cases in which the removal of some definite cause was followed by a cure. It is probable that continued reports of this character will gradually enlarge our knowledge of the relative importance of the various etiological factors which are concerned in this disturbance of function.

VESICAL CALCULUS

The frequency of occurrence of stone in the bladder varies greatly in different parts of the world. The condition is apparently commoner in England, France, and Russia, than in Germany. There is an area along the lower Danube where it is particularly common. It is comparatively rare in the United States. The condition is always relatively less common in children than in adults, and it is very rarely encountered in this country. Bókay's statistics show that the disease is seen more often in boys than in girls, and is seen most often between the ages of two and seven years.

ETIOLOGY.—Interference with the urinary discharge is regarded as the most important factor in etiology, and the frequency of phimosis is believed to explain the usual occurrence of the condition in boys. It is probable that the immediate basis of stone formation is a concretion descending from the kidney.

PATHOLOGICAL ANATOMY.—The calculus may be composed of uric acid and its salts, of phosphates, of oxalates, or of a combination of these salts. The uric acid stone is the commonest. The irritation of the calculus gives rise to a catarrhal inflammation of the mucous membrane of the bladder.

SYMPTOMS.—The first symptoms are increased frequency of micturition, and pain on micturition. There is often diurnal incontinence of urine. The pain occurs at the close of micturition, and is usually referred to the end of the penis, to the perineum, or to the groin. There may be a sudden stoppage in the flow of urine, with vesical tenesmus. The straining causes rectal tenesmus, and may lead to prolapse of the rectum, which is a fairly common complication. Gradually signs of catarrhal inflammation of the bladder appear, but they are usually less marked in children than in adults. The urine in most cases shows no more than mucus and a few pus cells; some red blood corpuscles may be present, but marked hematuria is rare. The irritation may lead to masturbation.

DIAGNOSIS.—Three methods of investigation are available in making a diagnosis: These are rectal examination, Roentgen-ray examination, and exploration with a sound. The stone may often be felt on bimanual examination with one finger in the rectum, but a negative finding does not exclude calculus. A roentgenogram should be taken in all suspected cases; a positive plate is diagnostic, but a negative plate is inconclusive. When both these methods are negative, the bladder should be explored by means of the sound.

PROGNOSIS.—This depends upon the treatment, and upon how soon the condition is recognized. It should be favorable if the case is treated before a severe cystitis has taken place.

TREATMENT.—The treatment is purely surgical.

IV. INFECTIONS

ACUTE CYSTITIS

Acute cystitis is not a common affection in infancy and childhood.

ETIOLOGY.—It may be caused by a vesical calculus, by irritants, such as turpentine, and also occasionally by the extension of infection through the genital tract, but this is less common than in adults. It is not an infrequent complication of typhoid fever and many other diseases from direct infection by the bacteria which are eliminated by the urine. Gonorrhea and infections by the pyogenic organisms are direct causes of acute cystitis. It is more common in girls than in boys.

SYMPTOMS.—In children the chief symptom is frequent and painful micturition. This local symptom is usually accompanied by fever, which may be high, and by general symptoms of malaise, fretfulness, and crying from vesical pain and tenesmus. The urine is passed in small quantities, and, as a rule, is of a reddish color at first, and gradually becomes of a lighter color. The specific gravity is high. When freshly passed it is acid, but it quickly becomes alkaline; there is a heavy sediment and a trace of albumin. Microscopic examination shows chiefly pus in large quantities, squamous epithelium, and some blood. To establish the diagnosis in females it is sometimes necessary to examine a catheter specimen. In infants the symptoms are so mild that the condition is often unsuspected.

PROGNOSIS.—The prognosis of acute cystitis is good after the removal of the cause.

TREATMENT.—The especial cause of the attack must be looked for, and removed if possible. The child should be kept perfectly quiet in bed, and should be made to drink a great deal of water. The diet should be chiefly milk. Hexamethylenamin should be given in doses proportioned to the age of the child, and also sodium benzoate or acid sodium phosphate in doses sufficient to insure acidity of the urine. The bromides are useful sedatives in the symptomatic treatment.

CHRONIC CYSTITIS

ETIOLOGY.—Chronic cystitis may be caused in children, as in adults, by a vesical calculus, by foreign bodies in the bladder, by tumors, by papillomata, and by tuberculosis.

SYMPTOMS.—Micturition is frequent and at times painful. Later there may be a constant dribbling of urine, giving rise to an offensive ammoniacal odor and causing irritation about the genitals. In addition to these local symptoms there are general symptoms of anemia and loss of weight. The urine is ammoniacal, offensive in odor, and turbid, has a heavy ropy sediment, and contains a trace of albumin. The sediment should be examined as soon as possible after the urine is passed, because the ammonia which is produced from the urea disintegrates the cells. The examination will show a large quantity of pus, some blood, bladder epithelium, and crystals of triple phosphate and urate of ammonium. Tubercle bacilli may be found in the urine in tuberculosis of the bladder, but a prolonged and careful search is often necessary.

PROGNOSIS.—The prognosis of chronic cystitis depends upon the cause, upon the length of time during which the disease has persisted, and the presence or absence of a secondary infection of the kidney.

TREATMENT.—The urine should be diluted by giving distilled water in large amount. The treatment with hexamethylenamin and sodium benzoate should be employed as in acute cystitis. Washing out the bladder with weak solutions of permanganate of potash, or boracic acid, is of use in many cases, and local applications may be made in tuberculosis of the organ. Operative treatment is indicated when a calculus is causing the disturbance.

C. DISEASES OF THE GENITALS

I. MALFORMATIONS

ADHERENT PREPUCE.—In early life there appears to be a physiological adhesion of the prepuce to the glans penis. As the child grows older these adhesions normally disappear. When the adhesion between the prepuce and the glans remains permanent, the condition gives rise to various symptoms of nervous irritation. In such cases the prepuce should be retracted, adhesions should be broken up so as wholly to expose the glans, the smegma should be washed away, and the glans anointed with vaselin, and the prepuce drawn back over the glans. Care must be taken not to leave the prepuce retracted too long, as drawing it forward may become difficult, and if it is left retracted, paraphimosis will result. Drawing back, cleansing, and anointing should be repeated daily, until there is no tendency for the adhesion to recur.

PHIMOSIS.—In this condition the prepuce is so narrow that it cannot be retracted over the glans. Cases have been seen in which the prepuce had no opening. In other cases the opening is so narrow that the flow of urine is interfered with. Still more commonly the orifice, while sufficiently narrow to prevent retraction, is not so small as to interfere with the flow of urine. The prepuce is also frequently greatly elongated, and this together with the impossibility of retraction favors the accumulation of smegma about the corona. In some cases the prepuce can be retracted, but it is so elongated that it is impossible to keep the parts clean.

The impossibility of cleanliness in phimosis favors infection, and balanitis is a common result. There may be urethritis and even cystitis. The irritation from retained smegma and from infection is a source of reflex irritation which may affect the entire nervous system. All sorts of reflex phenomena have been ascribed to phimosis. Nevertheless, in my opinion, the connection between these reflex phenomena and the condition of the penis is not so certain as many writers would lead one to suppose. Phimosis is certainly one of the etiological factors in enuresis and masturbation, but its importance as an etiological factor in other neuroses is somewhat doubtful. Many cases of marked phimosis are seen without reflex phenomena, but the possibility of phimosis as a contributing cause in a functional disturbance of the nervous system must be considered. Nevertheless, in my experience, when circumcision has been undertaken as a cure for various nervous conditions in boyhood, the results have been as a whole rather disappointing. Another result of the straining caused by phimosis is hernia.

TREATMENT.—In all cases of phimosis which give rise to symptoms of irritation, local treatment is indicated, and may consist in dilatation, incision, or circumcision; the latter is the most radical and produces the best results of complete relief from the morbid condition. In all cases, even if the phimosis is very slight, mechanical interference should be persisted in until absolute cleanliness can be secured, for in this way only will entire relief from the local and reflex symptoms be obtained.

HYPOSPADIAS.—The malformation known as *hypospadias* is the result of an arrest of development in the formation of the urethra and of the corpus spongiosum. The urethral groove should normally be converted into a canal by the growth and joining together of its sides. This process begins at the base and extends to the end of the penis. By an interruption of this process the urethra may be brought to an end and open at any point between the penoscrotal angle and the base of the glans. In the most common forms of hypospadias the glans alone is imperforate.

TREATMENT.—The treatment is wholly by plastic operation, and it requires the most delicate surgery to obtain a good result in the face of the many serious obstacles that this malformation presents.

EPISPADIAS.—The malformation of *epispadias*, in which the urethral canal opens upon the dorsum of the penis, is still more difficult to deal with than is hypospadias. It is commonly associated with extroversion of the bladder, and is very rare.

TREATMENT.—A partial plastic operation and the wearing of some form of urinal constitute about all that can be done for these cases.

UNDESCENDED TESTICLE. CRYPTORCHIDISM.—The testicle should descend into the scrotum at about the eighth month of intra-uterine life. In certain cases it does not descend, and if the descent does not take place within the first few years of life its function is lost from atrophy. It is, therefore, important in those cases in which the testicle descends and returns to the abdominal cavity to retain it in the scrotum by means of apparatus. Operation for this condition is not often successful. At times an undescended testicle is found in combination with an inguinal hernia.

HYDROCELE.—Several anatomical varieties are met with in hydrocele, as in hernia. Thus, if the collection of fluid occupies a freely open funicular process, we have the *congenital variety*, and the fluid can easily be returned to the abdominal cavity by placing the child on its back and elevating the scrotum. This is true also of *funicular hydrocele,* in which the fluid occupies an open funicular process, but is bounded below at the point where the tunica vaginalis has become walled off, leaving the testicle in a separate compartment underneath. When the funicular process has become walled off from the abdomen, but is still in communication with the tunica vaginalis, there may be a collection of fluid, which is then known as an *infantile hydrocele;* in this form the fluid is irreducible. True hydrocele of the tunica vaginalis may be met with in children as well as in adults, but is rare.

Encysted Hydrocele of the Cord.—There is another form of hydrocele which often escapes recognition, but perhaps still oftener is diagnosed as hernia and treated with a truss. This is the encysted hydrocele of the cord. If in the course of the spermatic cord a hard, rounded swelling appears, and the testicle is found in its proper position in the scrotum and the inguinal ring clear, one is very surely dealing with a hydrocele of this kind. Having made the diagnosis, it should be evacuated with a fine aspirating needle. About 4 c.c. (1 drachm) of clear straw-colored fluid is generally drawn off, and the tumor usually disappears.

Encysted Hydrocele of the Canal of Nuck.—Analogous to hydrocele of the cord in boys is an accumulation of fluid in the canal of Nuck in girls. The appearance of the swelling is the same in both cases, and the treatment should be the same.

Treatment.—The treatment of all forms of irreducible hydrocele is first by aseptic evacuation of the fluid with a fine canula and trocar, or by an aspirating needle. If this, after repeated trials, fails to effect a cure, extirpation of the sac is the only sure method, although the injection of a weak solution of iodin is highly recommended by many authors. It is, however, dangerous in children, as the occasional connection of the hydrocele sac with the abdomen is not to be forgotten. Reducible forms of hydrocele are generally to be treated by a truss, in the same manner as herniae, to try to effect a closure of the neck of the canal. If this is successful they can then be treated in the ordinary way. The outlook, however, is poor, and such treatment is generally unsatisfactory.

MALFORMATIONS OF THE FEMALE GENITALS.—Various developmental malformations in the female genitals are encountered as rare congenital anomalies. Their detailed description is not of much practical value. Their nature is usually easily recognizable, and the only practical question which arises is whether or not they can be benefited by surgical interference.

II. NEW GROWTHS

Neoplasms involving the genital organs are rare in early life. Tumors of the testicles may be found in infancy and early childhood. These may be congenital or acquired. The congenital tumors are very rare, and are usually of the dermoid variety. The most common of the acquired tumors are carcinomata and sarcomata, which are very malignant. The rapid growth and the large size of this variety usually render the diagnosis easy.

The only benign tumor of the female genitals which is common enough in children to be noted, are the vaginal polypi which are characterized by hemorrhage. Malignant new growths of the genital organs are commoner in girls than in boys. Most of the neoplasms found in early life appear to be congenital. The vagina is the most common site, and the new growth is usually a sarcoma of a very malignant kind. The uterus may be affected both by sarcoma and carcinoma. Malignant tumors of the ovary are rare.

The diagnosis of these new growths presents no special features in early life. The only treatment is surgical, and the prognosis of malignant tumors is extremely bad.

III. FUNCTIONAL DISTURBANCES

MASTURBATION

Masturbation may be defined as a habit of exciting pleasurable sensations in the genital organs by means of mechanical excitation. It is not uncommon in early life, even in infancy. The masturbation of infancy and early childhood has been described as "pseudomasturbation," the distinction being that at this age the sexual orgasm characteristic of true masturbation cannot take place owing to the lack of development of the internal genitals. While this is true of the masturbation of early life, under the broader definition of masturbation given above, the distinction in terminology is unnecessary.

Masturbation is seen in children of all ages. It is not uncommon in infants, many cases having been observed during the first year, even as early as the sixth or seventh month. The great majority of the cases of the "pseudomasturbation" of infancy and early childhood occur in girls. The true masturbation of late childhood is much more common in boys.

ETIOLOGY.—It is easy to understand the pleasurable sensation from the orgasm obtained in older children by masturbation. In infants and young children with undeveloped internal sexual organs, the explanation of masturbation is less obvious. Rachford's explanation of this phenomenon is the most plausible. The nerve supply of the external genitals is closely connected with that of the internal genitals. The more fully developed external genitals are capable of responding to reflex excitation, and "this excitation finds expression in producing a miniature syndrome so like true masturbation that one must conclude that this portion of the infantile genital system, which is later in life to come in closer touch with the fully developed internal sexual organs, must even at this early date in its development have impressed upon it the peculiar physiological function which makes it respond to reflex excitation by mimicking the sexual orgasm." (Rachford.)

The etiological factors which lead to the practice of masturbation are somewhat similar to those seen in the neuroses, namely, *local sources of irritation*, and *general neurotic hyperirritability*. It is probable that in infancy and early childhood the cause of the first practice of mechanical excitation is usually some local irritation. Among such sources may be numbered vulvovaginitis, adherent clitoris, eczema of the labia, adherent prepuce, phimosis, balanitis, pinworms, an acid irritating urine and tight clothing. Any irritation causing the child to rub the external genitals may be the first cause. Certain forms of play or exercise may involve a rubbing of these parts.

The infant having once produced a pleasurable sensation by mechanical excitation, the practice develops into a *habit.* Whether or not the habit develops depends upon the degree of reflex excitation produced. This in turn is dependent upon the other factor in etiology,—the condition of the nervous system of the individual child. Hyper-irritability of the nervous system to reflex stimulation may be due to hereditary or to external factors. In many cases there is a neuropathic inheritance. Masturbation may be an early sign of some condition of mental deficiency, other stigmata of mental or moral degeneration appearing later. Among the external factors are the various faults in hygiene and environment which interfere with normal development, and tend to produce functional disturbances of the nervous system. Anemia, malnutrition, and a faulty hygiene of the nervous system are potent factors.

Once the habit is formed, it becomes in itself the most important etiological factor. Such powerful pleasurable sensations can be produced in no other way, and the frequent excitation of the nervous system makes it more and more irritable to less and less excitation, so that a fully developed vicious circle is produced.

In older children, the habit is most often learned through the imitation of other children. It is sometimes taught by a vicious nurse. It is often acquired accidentally through handling of the parts, such handling being due to a local source of irritation, or to a mere vagary in which the child takes its external genitals for a plaything.

In children over the age of ten years, the beginning development of the internal sexual organs causes the sensations accompanying masturbation to be more intense, and to resemble more closely those produced by the sexual act. Such cases resemble true masturbation in everything but the occurrence of an orgasm. In some cases a premature development of the internal sexual organs may make true masturbation a possibility during the later years of childhood, sometimes even before the tenth year.

SYMPTOMS.—In infants and young children masturbation is most commonly accomplished by what is called "thigh friction." The child lies usually upon its back with the thighs flexed, crossed, and pressed rigidly together. In this position it makes an up and down wriggling movement of the body while rubbing its thighs together. In other instances the genitals are rubbed with the hands or feet, or are rubbed against a pillow, piece of furniture, or some other foreign object. There is a great variety of ways in which the rubbing can be accomplished. The movements are accompanied by evidences of pleasurable excitement, such as flushing of the face, an intent expression of the eyes, and a general condition of nervous tension. The rubbing lasts only a few minutes, and is succeeded by general relaxation, some perspiration, and an appearance of quiet contentment. These attacks may be repeated many times a day, or intervals of days or weeks may elapse between them.

The nature of infantile masturbation is often unsuspected by the child's mother for a long time. The attacks are noticed, but are considered to be some "queer trick" without significance. In some cases the mother has a suspicion of the true nature of the condition, but regards such a thing as so impossible or unusual at so early an age, that she hesitates to tell the physician. In other cases the attacks are described by the mother as a "spasm."

In boys approaching the age of puberty, who have learned the habit by imitation, masturbation is usually accomplished by the hand, although pressing or rubbing the penis upon the bed is not uncommon. Older children, with a consciousness of doing something wrong, usually seek seclusion while practicing the habit. The habit is most likely to be practiced when children lie long awake in bed, either in the evening or in the morning. Conditions which produce a temporary or permanent deterioration of the general health tend to make the habit worse, and may bring about a relapse after the habit is supposedly cured.

A great variety of injurious effects upon the general health have been attributed to masturbation. As we have seen in considering etiology, the habit represents a vicious circle, in which causes and effects react upon one another. It is consequently difficult to say whether any clinical manifestations present are symptoms of the causes leading to masturbation, or symptoms of the effect of masturbation. The most common symptom seen in children who practice masturbation are excitability, "nervousness," loss of self-control, absent-mindedness, lack of power of concentration, loss of interest in amusements, mental depression and moral deterioration. All these symptoms are not present in each case, but some of them are usually found whenever the habit is practiced excessively. When the practice is begun at an early age, both mental and physical development may be notably interfered with. It is often stated that masturbation is a cause of insanity, epilepsy, and hysteria. I am very doubtful whether this statement is true as regards the sequence of cause and effect, on account of the relative frequency of excessive masturbation in early life, and the relative infrequency of the development of insanity, epilepsy or hysteria. When these conditions apparently follow masturbation, I believe it to be more likely that the masturbation is the first manifestation of a developing insanity, or of the neuropathic constitution leading to the development of epilepsy or hysteria.

Local signs of masturbation are more often absent than present.

There may be slight redness and swelling of the prepuce in boys, and of the labia or clitoris in girls. The organs may be abnormally large, or much relaxed. There may be a catarrhal vaginitis. In boys it is a suspicious sign if erection of the penis occurs as soon as the parts are touched for examination.

DIAGNOSIS.—While the first statements of the mother may be misleading, cross-questioning by the physician will usually elicit a description of the attacks sufficiently accurate for a diagnosis.

PROGNOSIS.—In general, the prognosis is good. The ease with which the habit can be eradicated varies with the age of the child, with the underlying causes, and with the length of time the habit has been practiced. The younger the child, the more favorable is the outlook. The pseudomasturbation of infancy tends toward spontaneous recovery in the course of time, and under treatment a permanent cure should be attained well within two years at most. In older children in whom either the habit has existed a long time, or who have reached an age when the sensation produced closely resembles that of the orgasm seen in the true masturbation of adolescence, the condition is much more difficult to deal with, and calls for time and patience. The tendency toward relapse is very strong.

As I stated above, I do not believe that masturbation, at least the pseudomasturbation of early life, leads to insanity or epilepsy. When masturbation is a symptom of degeneration, however, the outlook for a cure is very bad.

TREATMENT.—Masturbation is a habit neurosis, and consequently the most important indication in treatment is to *interrupt the habit*. This should be done as soon as possible, because whatever the original exciting cause, the condition has become a vicious circle which must be broken before the habit can be cured.

If infants and young children could be watched every minute of the day and night, and the practice of the habit forcibly prevented as soon as begun, the habit could be broken in this way. It is usually impossible practically to carry out such close supervision; the infant is likely to begin the practice at any time when she wakes, and she cannot usually be watched every minute of the night. Therefore at night the use of some kind of mechanical restraint is indicated, the mechanism depending upon the manner of masturbating. If thigh friction is practiced, the thighs may be fastened to the sides of the crib in such a way as to prevent them from being brought together. The heels of pajamas fastened to the mattress may accomplish the same purpose. In very young infants a large diaper may be folded in such a way as to prevent approximation of the thighs. In very obstinate cases, double side-splints may be used, or some other mechanical device which accomplishes the same purpose. If masturbation is practiced with the hands or feet, they should be tied so as not to be able to reach the genitals.

During the day, or at any time when mechanical restraint is not in use, the practice of the habit must be forcibly prevented by constant watching. The child should be kept in a sitting posture as much as possible, as the habit is most often practiced when the child is lying down.

Some writers state that punishment is effective in older children. In my opinion it is just the other way. In infants, and children too young to reason, corporal punishment, if carried out only at the time when the child is caught practicing the act, may be very effective. Children at this age are like puppies, and can be trained by the same methods. In children over three years of age, punishment, whether corporal or not, is ineffective and inadvisable. The mother should talk to the child about the condition in a thoroughly matter-of-fact way, and should make every effort to gain the child's confidence. She should avoid giving the child the impression that the habit is essentially wicked or immoral, but should lay stress only on its physical aspect, representing it as wrong only because of its effect on the health. She should direct her influence toward strengthening the child's will and self-control, and should try to bring about such relations with the child that it will always gladly report each lapse. She should avoid an attitude which is likely to lead the child to the practice of lying and concealment. This psychic treatment is very important in older children. Sometimes a complete change of surroundings is useful, the child being put in the charge of some competent person who has hitherto been a stranger to it. Removal of older children from bad companions is always essential.

As soon as these measures directed at the interruption of the habit have been planned, the physician should attend to the removal of all possible sources of irritation. Such conditions as adhesions of the prepuce or clitoris, phimosis, eczema of the genitals, pinworms, diseases of the rectum, ill-fitting clothing, and indeed every possible source of local irritation, should be removed. Some writers believe that increased acidity of the urine is a common local cause of reflex irritation in these cases. The urine should be tested, and if concentrated and highly acid, plenty of water should be given, and appropriate doses of potassium acetate or potassium citrate.

Finally, every effort should be made to improve the child's general condition. This always means a rigid scrutiny of every detail of the hygienic surroundings. All faults in the patient's mode of life should be corrected. The diet should be carefully regulated. Cold baths are often of value. Tonics, such as iron, arsenic, or cod liver oil, have their place at times. In short, every effort should be made to build up the general health and favor rugged normal development.

IV. INFECTIONS

VULVOVAGINITIS

This is an exceedingly common affection in early life.

ETIOLOGY. The Microörganism.—The great majority of cases of vulvovaginitis in early life are due to infection with the gonococcus of Neisser. The organism seen in these cases has the same morphological and cultural characteristics as has the organism causing the gonorrhea of adults. The question whether the organism found in the very common vulvovaginitis of little girls represents a strain of the gonococcus differing from that found in ordinary gonorrhea, will be discussed under Problems and Research.

A certain number of cases of vulvovaginitis in early life are seen, in which the gonococcus is not found. In these cases the inflammation is of a catarrhal type, and the cause is either a mixed infection with organisms of comparatively low virulence, or some irritant such as dirt.

Transmission.—The disease is a contact infection, the mode of transmission being similar to that of such diseases as scarlet fever and diphtheria. It is very highly contagious. The ultimate source of the infection of children is probably the venereal gonorrhea of adults. Direct contact occurs when little children sleep with a mother or sister suffering from the disease. Under such conditions it is easy to understand how the contaminated discharges of the adult may be brought in contact with the vulva of the child. Many cases have been traced to this source. Infection in the home also very often takes place by indirect contact. The discharges of the adult may not only contaminate clothing, towels, baths, toilet-seats and similar carriers, but the hands of the adult, contaminated by the discharges, may contaminate any object with which they come in contact. The child may contaminate her hands from those of the mother, or from these various objects, and may then transfer the infection to her vulva.

Outside the home, the contact infection can spread from child to child. It is particularly likely to spread in institutions, where infants and young children are under more or less crowded conditions. It is an especial scourge of infants' hospitals and of homes and asylums for infants and children. The most common medium of spreading in institutions is the diapers. Other carriers are thermometers, towels, sponges, wash-cloths, bath-tubs, bath-water, clothing, bed linen, syringes and so forth. At times a hospital ward epidemic is very difficult to stop. It is probable that in such epidemics the most important medium of transmission is the hands of the nurses, which easily become contaminated, and which may contaminate every object with which they come in contact. The hands of other nurses, who have the care of unaffected children, can then become contaminated from these objects.

Outside of institutions, the infection can be communicated very easily from one child to another. The habits and mode of life of children are such as to favor the spread of a contact infection of this kind. Children are brought into close contact in schools, in public places, and in play. Children are very apt to rub or handle the genitals, even when not victims of the habit of masturbation, which is very common in children suffering from vulvovaginitis. Thus children contaminate the hands, and communicate the infection either directly or through contamination of objects to the hands of other children, which in turn convey the infection to the vulva. In schools and public places the disease may also undoubtedly be spread by the toilet seat. The statistical reports of some writers suggest that the majority of children are infected from adults in the home, while those of other writers suggest that communication from child to child is the most common source of infection. Criminal assault is a very rare cause in children.

PREDISPOSITION.—The vaginal mucous membrane of little girls is extremely susceptible to gonococcus infection. Only this fact can account for the relative frequency of gonorrhea as a non-venereal contact infection at this age. It is probable that whenever gonococci are conveyed to the external female genitals, infection takes place. This extreme susceptibility is probably to be explained by the relatively small size of the labia at this period of life, and the consequent inadequate protection of the underlying mucous membrane.

The urethra of boys is not susceptible, and gonococcus urethritis is extremely rare in early life.

The disease is commonest among children who live under the crowded conditions of the tenements. It is, however, not very rare among the children of the well-to-do.

SYMPTOMS.—There are usually no subjective symptoms of any kind, and the disease is discovered accidentally. The only sign of the disease is the *vaginal discharge*. The amount and character of the discharge vary with the severity of the case. Most commonly it is sufficiently profuse to soil the napkins, underclothing, and bed linen, and has a mucopurulent appearance. In very mild cases, it is glairy and tenacious, and so small in amount as to be almost unrecognizable. In severe cases it is thin and purulent, very profuse, and of a yellowish or greenish color, at times tinged with blood. It may cause excoriation of the labia and inner surface of the thighs. There may be frequent and painful micturition, owing to involvement of the urethra.

Examination of the vulva often shows very little signs of inflammation, especially in infants. At times there may be marked redness with swelling of the labia. If a small speculum be introduced, examination will show that the entire mucous membrane of the vagina is involved in the inflammation, which varies in intensity. In infants and young children the inflammation is usually confined to the hymen, vaginal mucous membrane, and possibly the urethra. In older children, the cervix uteri is usually involved, and examination with the speculum will show purulent discharge coming from the cervix, and may show erosions. In very severe cases in older girls, the entire endometrium and even the Fallopian tubes may occasionally be involved. In these very severe cases there may be fever and constitutional symptoms as in adults.

COMPLICATIONS.—Complications are very rare in children as compared with adults. They are particularly uncommon in infants and young children. One of the surprising features of the disease is the rarity of conjunctivitis. The ease with which the disease is transmitted from the vaginal mucous membrane of one child to that of another suggests that the infection must frequently be carried to the eyes, and the rarity of conjunctivitis can only be explained on the basis of an insusceptibility of the conjunctiva in early life, or of a difference in the strain of gonococcus which causes the disease. Swelling and suppuration of the inguinal lymphnodes are also very rare. Arthritis is occasionally seen, usually in older children, rarely even in infants. Other still rarer complications mentioned in the literature are pelvic peritonitis, endo- and pericarditis, cystitis, proctitis, and meningitis.

DIAGNOSIS.—The diagnosis depends upon the microscopic examination of the discharge. In every child with a vaginal discharge, no matter how slight, such an examination should be made. An abundant purulent discharge is always due to the gonococcus. The slighter mucoid discharges are usually due to gonococcus infection, but may be of simple catarrhal origin. In ordinary cases which have not been treated, the gonococci are found in large numbers. In exceptionally mild cases, and in cases which for some time have been under treatment, the gonococci are often very difficult to demonstrate. The examination should be considered positive whenever any Gram negative diplococci are found within the leukocytes. If Gram negative diplococci are present, but are not found in the cells, the examination should be called suspicious, but for purposes of prophylaxis such a case should be considered positive. The examination should be repeated in such cases until a frankly positive result, or a number of negative results are obtained. The examination is negative when no Gram negative cocci can be found.

The smear should be taken with a sterile cotton swab, or with a platinum loop. In cases with a profuse purulent discharge, the material can be taken from the pus revealed when the labia are separated. When the discharge is slight, or when the material collected outside the vagina gives a negative finding, material should be obtained from the mucous membrane of the vagina itself by means of a small speculum, such as a female urethroscope.

Cases have been reported in which the examination of smears was repeatedly negative, but in which the presence of gonococci was demonstrated by means of cultures. Another means of diagnosis is the complement fixation test. This latter test is considered to be of especial value in establishing the fact of permanent cure. The final value of the complement fixation test in the diagnosis of gonococcus vaginitis in early life has not been settled. Both cultures and the complement fixation test require an expert bacteriologist, and a thoroughly equipped bacteriological laboratory, and are more available in hospitals than in ordinary practice.

PROGNOSIS.—The disease has an ultimately favorable prognosis. There is no danger to life, and in the majority of cases there is little if any deleterious influence upon the general health. The only serious possibility to be apprehended is that of a complication, and these are rare. The majority of cases eventually recover completely.

The outlook as to the rapidity with which a cure may be obtained is not so favorable. The course of the disease is usually long and tedious. In the most favorable cases from one to four months of treatment are required. The profuse vaginal discharge will under treatment rapidly diminish and disappear, but a very slight, almost imperceptible discharge will continue, and gonococci will still be demonstrable at times on microscopic examination. In many cases the disease persists for many months or even years, in spite of treatment. Relapses are common. No case should be considered as cured until repeated negative microscopic examinations have been obtained.

PROPHYLAXIS.—The problem of prophylaxis in the gonococcus vaginitis of children has been the subject of much discussion in recent years. As the original source of the spread of the infection among children is venereal gonorrhea in the adult, prophylactic measures, to be effective, should begin in the home. Education of the public is of the first importance. In general, the public is unaware of the extreme frequency of the disease in children, and of the fact that in them it is not a venereal disease, but that it is an extremely contagious infection acquired in the same way as are other contact infections. The public should be instructed on these points. The idea that vaginitis in young girls is a venereal disease has been a very harmful one, both because it has prevented knowledge of the real way by which the disease is acquired, and because it has led to cases being concealed instead of being brought to the physician. Not only should nurses, social workers, and the authorities of institutions and schools be thoroughly acquainted with the frequency and nature of the disease and with its mode of transmission in children, but mothers should also be instructed. Even young girls themselves, when old enough to understand intelligently, should be instructed as to the dangers of infection, and the means of its avoidance. Such education of the public can only be carried on through the agency of physicians, and of the various social service organizations. In many cities, the most valuable work in the prophylaxis of gonococcus vaginitis in the home is being carried out by the medical social service departments of some of the large hospitals, and by the establishment of medical social service centers throughout the city.

The measures of prevention which should be employed in the home are those which naturally apply to the particular mode of transmission of the disease. It is essential that the infected person should sleep alone, and that her sheets and clothing should be kept from contact with and be washed separately from those of the rest of the household. She should wear a vulvar pad of some material that can be burnt after use. She should be taught the importance of proper cleansing of the genitalia, the danger of contaminating the hands and of transmitting the infection to other persons and objects, and the necessity of washing the hands after handling her clothing, or after any possible contact with the discharge. The greatest care about bath-water, and the cleansing of bath-tubs and toilet-seats, should be secured.

In the schools the chief means of transmission is probably the common toilet-seat. No other form than the U-shaped toilet-seat should be used in the schools and in all other public places. The question has been raised whether the disease should be made reportable to the health authorities, with exclusion of infected children from the public schools. This would involve the examination of smears from all the girls attending school. It is the only means by which thorough prophylaxis in the schools could be obtained, as there are many opportunities for transmission besides toilet-seats. There are similar opportunities for transmission outside the schools, in the streets and playgrounds, and really effective prophylaxis would demand not only exclusion from school, but also the visiting of homes by the health authorities, and the quarantine of infected households. Opinions have differed as to the advisability of the public health authorities taking up the prophylaxis of vaginitis in this way. In my opinion such extremely radical measures are not justified by the severity of the disease.

In institutions the problem of prevention is very difficult, but can be overcome by the most careful attention to every detail. In the Infants' Hospital there have been no cases for two years, and only four three years ago. The examination of a vaginal smear from each female candidate for admission should be a matter of routine, and no case with a positive or suspicious smear should be admitted, unless the hospital has a department especially devoted to the care of such cases. Children with any vaginal discharge but a negative smear should either not be admitted, or should be kept under the strictest quarantine until repeated negative smears have been obtained.

The nurses should watch all female patients for the slightest appearance of vaginal discharge, and should report immediately, in order that smears may be taken. In some institutions routine smears from all female patients are taken weekly. Upon the discovery of a case of infection, the child should be isolated under a quarantine fully as strict as that employed in scarlet fever or diphtheria. As far as the danger of spreading is concerned, I would far rather have a case of scarlet fever get into my wards than one of vulvovaginitis. It is important that not only the patients be quarantined, but also all nurses and attendants coming in contact with those patients. They should come into no contact with other patients, nor with other nurses. They should be instructed as to the nature of the disease, and as to the danger of carrying the infection on their hands and producing conjunctivitis or the reinoculation of convalescents. Their clothing should be kept separate and should be disinfected with the same care as would be employed if the disease were smallpox. Infected patients should wear inside the diaper a pad which is burned after use. Each infected child should have its own thermometer, toilet articles, and eating utensils. Indeed, in any hospital in which rectal thermometers are used, it is not enough to keep the thermometer in some antiseptic solution, but each child should have its own thermometer.

TREATMENT.—The therapeutic procedures which are employed in the vulvovaginitis of early life are *irrigations*, *instillations*, *local applications*, and *vaccines*.

IRRIGATIONS.—The child is placed on her back on a table, with her hips raised by a bed-pan. The tube of a fountain syringe is connected with a soft rubber catheter, size 12 or 14 French scale, by means of a bit of glass tubing. The syringe filled with the irrigating solution heated to 106 F. is hung about two feet above the patient. The tip of the catheter, well lubricated, is introduced through the opening in the hymen as far as it will go. From one to two quarts of the irrigating solution are allowed to run into the vagina, passing out around the catheter. The solutions used for irrigating are boric acid, 2 per cent; sodium bicarbonate, one teaspoonful to a quart; potassium permanganate, 1-8000 or 1-10,000; bichlorid of mercury, 1-5000; Lugol's solution of iodin, 1-1000. The two first, boric acid and sodium bicarbonate, are best in the more acute stages of the disease, the sodium bicarbonate solution being especially soothing. Later, the potassium permanganate solution is best for routine use. The use of the bichlorid and Lugol's solution, and of a stronger, 1-1000, permanganate solution, is only indicated when irrigation is the only method of treatment employed. When, as is preferable, irrigations are only preparatory to instillations or local applications, the first three solutions only should be used.

INSTILLATIONS.—After the irrigation is completed, the catheter is separated from the glass connection, remaining in place, and one or two drachms of a solution of one of the silver salts is injected through the catheter by means of a hand syringe. The catheter is then withdrawn, and the child is made to hold her thighs together to prevent the escape of the solution, and lies in this position with her hips still elevated for fifteen minutes. In using these instillations, it is best to begin with a 10 per cent solution of argyrol. Later the strength of the solution may be increased to 20 per cent, or a 5 per cent solution of protargol may be used.

APPLICATIONS.—With the child in the same position, after cleansing the external genitals, an electric endoscope, such as the Kelly, is passed into the vagina. The size of the endoscope should depend upon the opening in the hymen, not upon the age of the child; it is essential that the introduction of the instrument should cause no pain. The obturator of the endoscope is withdrawn, the light inserted, and the tube withdrawn about a quarter of an inch, until the cervix presents. Any secretion is removed with an applicator, and then a swab is moistened with the solution, and applied to the cervical canal. Then, as the endoscope is slowly withdrawn, the solution is applied to the vaginal walls. The solutions used are nitrate of silver, 10 per cent, Lugol's solution of iodin, and protargol. Perrin claims remarkably good results from a solution consisting of protargol, 8 grammes, distilled water, 8 grammes, glycerin to 50 grammes. Applications are useful in the most chronic and persistent cases.

VACCINES.—Vaccine therapy has been extensively tried in the gonococcus vaginitis of early life. The results have been disappointing on the whole, although there is much difference of opinion as to the value of this form of treatment. Many writers have reported excellent results, while others have reported no evidence of benefit. My own experience with vaccine therapy has not been favorable. It is evident that if there is any effect, it is very uncertain. There is more evidence in favor of the value of vaccine therapy in the complications, particularly in arthritis. There is no evidence that autogenous vaccines have any advantage over stock preparations. The dosage is still wholly empirical. It is customary to begin with doses of about 50,000,000, and increase to 75,000,000, and then to 100,000,000. The doses are repeated every five or six days. If no notable improvement is seen after six or eight injections, the further continuation of vaccine therapy is useless.

ROUTINE TREATMENT.—In acute cases, irrigation and instillation should be used. The irrigations should be given with one of the milder solutions, sodium bicarbonate or boric acid, and the instillations should be with 10 per cent argyrol. Later the potassium permanganate solution should be used in the irrigations, and a stronger argyrol solution in the instillations. The irrigation followed by instillation should preferably be given twice a day, but if this is impossible, a good result can often be obtained with one treatment daily. In chronic or very persistent cases, the irrigations should be continued, but applications, given three times a week by the method described above, should be substituted for the instillations. In very obstinate cases vaccine therapy may be tried.

This routine of treatment should be continued for at least a month after the disappearance of gonococci from the smears. A catarrhal discharge without gonococci can be prolonged by too persistent treatment. After treatment is stopped, frequent smears should be taken from the depths of the vagina, and if at any time the gonococcus reappears in the smears, treatment should be resumed. The question as to how long a period with negative smears should elapse before the patient is pronounced cured is an uncertain one. Some writers believe that the disease is practically an incurable one, and that the infection may remain latent for an indefinite period, and then again cause symptoms. It is true that the disease may remain latent for long periods, but nevertheless I believe that some of the apparent instances of recurrence after a long period of latency are really instances of reinfection. I believe that the disease, like the gonorrhea of adults, is a curable one. It is more difficult to say just when we should consider a case as cured. The required period of freedom from discharge and gonococci must be somewhat arbitrary. I believe that a case should be kept under the closest possible observation, with smears taken as frequently as possible, for at least four months after treatment is stopped. Even after this time it is advisable to take smears every one or two months for a considerably longer period.

Urethritis as an associated condition is less common in children than in adults. It may be treated by the instillation into the urethra of 1 or 2 c.c. of a 5 per cent argyrol solution.

PROBLEMS AND RESEARCH.—In the literature of the vulvovaginitis of early life, the following subjects are being discussed: (1) Methods of prophylaxis, especially the question of regulation by the public health authorities; (2) methods of treatment; (3) whether the organism is the same as that which produces the gonorrhea of adults, or whether it represents a different type or strain; (4) the value of the complement fixation test in diagnosis, and in confirming the fact of cure.

The question as to whether the organism found in the vulvovaginitis of children, is the same as that causing gonorrhea in adults, was suggested by certain peculiarities of the disease in early life. In children as compared with adults, the mildness of the local inflammation, the absence of constitutional symptoms, the rarity of complications, and the extreme transmissibility from child to child, suggested that the disease might be due to a different organism. The organism is exactly like that of adult gonorrhea in morphology and in cultural characteristics. Some evidence has been brought forward by research workers, notably by Pearce of the Rockefeller Institute, that the organisms of adult gonorrhea and juvenile vaginitis represent two distinct types. This evidence consists in the results of immunological tests—differences in agglutination and in complement fixation. It has not been fully confirmed. On the other hand, there is undoubted evidence of transmission of the infection from the adult to the child, and later from child to child, and this is strong evidence that the organisms in adults and children are identical. It is possible that the gonococcus, like Rosenow's streptococci, may be capable of acquiring different characteristics of virulence and different immunological reactions, when transplanted to the new environment of the juvenile vagina. Such a theory, while wholly unproven, would explain the conflicting evidence on this question.

The question of the value of the complement fixation test is of practical importance in connection with the question of the permanency of cure. This is still unsolved.

ORCHITIS

Orchitis, or inflammation of the testis proper, occasionally results from direct injury, much more rarely from infection in gonorrhea, syphilis, and tuberculosis. When present it is commonly accompanied by hydrocele. The orchitis which so commonly follows mumps in the adult is much less common in children. The treatment consists in support of the testicle by means of a suspensory or of a bandage, and in the application of cold by means of the ice bag or cold compresses.

EPIDIDYMITIS

Acute epididymitis may be caused by trauma or by any irritation of the mucous membrane of the urethra. In this disease the whole scrotum is apt to be hot and tender, and the child is in great pain. The epididymis is much enlarged and exquisitely tender, and pushes the testis forward. The cord is often implicated, becoming enlarged and painful on pressure.

The treatment should be energetic, as, owing to the swelling of the tissues about the testicle, there may be so much pressure that the gland will be seriously damaged, although the subsequent atrophy may not declare itself for a considerable time. The child should be kept upon his back in bed, the bowels freed with a cathartic, and a series of cold compresses kept upon the scrotum. In all inflammation of the testis or epididymis the scrotum should be placed in such a position that the lower end of the testicle points upward.

DIVISION XIII

DISEASES OF THE BLOOD, SPLEEN, LYMPHNODES, AND DUCTLESS GLANDS

I. DISEASES OF THE BLOOD

ANEMIA

GENERAL PATHOLOGY.—Anemia is a condition of diminution of the erythrocytes or of the hemoglobin in the blood. Such a diminution may be relative or absolute. Unfortunately there is no way of estimating in the living the total quantity of blood, nor the total number of red corpuscles and quantity of hemoglobin. Consequently we can only recognize a relative diminution in the red corpuscles and hemoglobin. It is known, however, that the mechanism for maintaining the volume of the blood is more perfect, and works more rapidly than that for maintaining the cellular content. It is the hemoglobin which of all the blood elements has the most important physiological function, namely, the carrying of oxygen. It is functional weakness of the blood which is of most importance as affecting the health of the individual. Consequently the recognition of a relative diminution of the hemoglobin is the most important factor in estimating the functional capacity of the blood.

PATHOGENESIS.—A relative diminution in the amount of hemoglobin, or in the number of red corpuscles which carry the hemoglobin, may take place in three ways:

1. By mechanical loss from the body.
2. By increased blood destruction.
3. By diminished blood formation.

The first way requires no detailed description. It explains the anemia which is seen after hemorrhage. In such a condition there is an immediate absolute diminution in the number of red corpuscles. The accompanying diminution in the volume of blood is so rapidly restored that there remains a relative diminution in the number of cellular elements.

The proportion of red corpuscles in the fluid blood is kept constant by the balance between blood formation and blood destruction. New red cells are constantly being formed by the bone marrow, and the old cells are constantly being destroyed, chiefly in the spleen. When any cause interferes with the function of blood formation, the result is either a diminution of the number of erythrocytes in the circulating blood, or else the formation of erythrocytes which are low in hemoglobin content. There are various injurious influences which may lead to destruction of the erythrocytes in the circulating blood, either in the spleen or elsewhere. Either of these two conditions, diminished blood formation or increased blood destruction, or both in combination, may account for the existence of a state of anemia.

ETIOLOGY.—The function of the blood is of such a nature that it affords every opportunity for the cells to be subjected to injurious influences. The causes of anemia are consequently many. Furthermore many of the causes are unknown. From what is known of the various conditions under which anemia occurs, can be deduced some facts as to the general nature of the causes which lead to impoverishment of the blood. The following are the principal general causes of anemia:

1. Developmental.
2. Myelopathic.
3. Mechanical.
4. Nutritional.
5. Toxic.

Developmental conditions always play a more important part in early life than in adult life. There is undoubtedly a condition of hypoplasia of the bone marrow which represents a condition of faulty or backward development, and which is probably always congenital. It is probable that this condition may be a contributing cause in the development of anemic conditions in infancy. A form of anemia has been described by some authors as a special type, in which this condition is the only or chief cause. This form of anemia has been described as *aplastic* or *a regeneratory anemia.*

Myelopathic anemia includes certain cases in which there is tumor formation involving the bone marrow, or sclerosis of the bone, in which conditions the anemia is apparently due to interference by the lesion with the function of the bone marrow. This is, however, a rare factor in the anemias of early life, but must be mentioned for the sake of completeness.

Anemia of *mechanical* origin requires no explanation. The cause is loss of blood from hemorrhage.

The pathogenesis of anemias of *nutritional* origin is diminution in the functioning capacity of the bone marrow. In most conditions of impaired nutrition all the important tissues of the body suffer to a greater or less extent. The bone marrow suffers with the other tissues, and the result is a functional insufficiency which leads either to a decreased formation of erythrocytes, or to the formation of erythrocytes deficient in hemoglobin content. It is probable that the various factors of hygiene and environment which exercise an influence upon nutrition may exercise an unequal influence in various parts of the body. Under some circumstances, for example, we know that it is the growth of the bone which is most affected, as in rickets. In other instances it is the development of adipose tissue. There are quite probably certain external factors which exercise a particularly unfavorable influence upon the nutrition and functional capacity of the blood-forming organs. The anemias seen in certain conditions of faulty hygienic surroundings and in cachectic states may be largely nutritional in origin, but the toxic factor, next to be described, can never be excluded in such conditions.

The *toxic* causes of anemia may be divided into three classes as follows:—(1) Exogenous chemical toxins; (2) endogenous toxins of metabolic origin; (3) endogenous toxins of parasitic origin. The exogenous toxins are the known chemical substances such as potassium chlorate, phenylhydrazin, nitrobenzol, etc., which have a recognized hemolytic action. It is known that many diseased conditions involve a disturbance of the metabolism, which leads to the formation of toxic substances and to their circulation in the blood. That these toxic substances may have at times a highly injurious influence upon the red blood corpuscles, shortening their lives and leading to their early destruction, is highly probable. In many of the diseased conditions in which such metabolic toxins represent a possible factor, it is impossible to exclude other factors, such as nutritional disturbance of the bone marrow, or the action of parasitic toxins. Nevertheless, toxins of metabolic origin must be included among the probable etiologic factors in anemia, and the very rapidly developing anemia which occurs in nephritis is probably best explained on this basis.

The association of anemia with many acute and chronic infections leaves no doubt of the importance of bacterial toxins as an etiologic factor. Obviously the association of anemia with the chronic infections might also be explained on nutritional grounds. The association of rapid impoverishment of the blood with acute infections, such as is notable in rheumatic fever, however, can only be explained on the basis of a hemolytic action of the bacterial poisons. The severe forms of anemia produced by some of the animal parasites, such as notably the dibothriocephalus latus, have long been recognized.

If the various causes which can produce anemia acted separately, each cause producing a corresponding type of blood impoverishment, the description of the anemias of early life would be much simplified. The association of the anemic condition with any definite cause is, however, always difficult to prove, and it is probable that in many cases a number of causes act conjointly in producing the impoverishment of the blood. All that can be done under the heading of Etiology is to indicate the principal causes which are probably concerned in the production of anemic conditions. Even when anemia is associated with some definite recognizable diseased condition, it is impossible in many cases to know the exact pathogenesis. Rickets, for example, is a disease which in early life is very frequently associated with anemia. In rickets a varied pathogenesis for the blood impoverishment is possible. The cause might be nutritional or toxic. We know that rickets is a nutritional disorder which affects all the tissues of the body, and it is quite possible that the anemia may be due to impaired nutrition of the bone marrow. On the other hand, rickets is a disturbance of the metabolism in which the formation of metabolic toxins cannot be excluded. In tuberculosis and syphilis we do not know definitely whether the accompanying anemia is nutritional or cachectic in origin, or is due to the effect of bacterial toxins. The same is true of the anemias which accompany gastro-intestinal disorders. Even if the cause of anemia is recognized as toxic, it is often impossible to know whether the toxin exercises its unfavorable influence upon the blood itself, causing increased destruction of erythrocytes, or whether it exercises its unfavorable influence upon the bone marrow, causing diminished formation of erythrocytes.

These conditions explain the great difficulty which has always been encountered by writers in presenting the subject of anemia. There are such great gaps in our knowledge that an adequate description of the pathology is practically impossible.

ANATOMICAL CHANGES.—It is easy to recognize a relative diminution of hemoglobin by colorimetric methods, and a relative diminution of the erythrocytes by counting methods. Such diminution is, however, not the only pathological condition of the blood found in anemic states. In some forms of anemia, the blood shows the presence of abnormal cells, such as nucleated erythrocytes (normoblasts or megaloblasts), erythrocytes of irregular size, shape, or staining reaction, (microcytes, macrocytes, poikilocytes, polychromatophiles), and abnormal white corpuscles (myelocytes). The presence of these cells is due to the effort on the part of the blood-forming organs to compensate for the loss by a hurried production of cellular elements. Loss from the blood of the essential red corpuscles acts as a stimulus to the blood-forming organs, and the result is the preparation of imperfectly developed cells. These cells are usually divided into embryonal and postembryonal types. The postembryonal types are cells which represent only an incomplete stage in the formation of the blood cells; they include microcytes, poikilocytes, polychromatophiles and normoblasts. The embryonal types are cells which are produced by an embryonal type of blood-forming tissue, and which are normally found only in the fetus; they include macrocytes, megaloblasts, and myelocytes. In the adult the presence of embryonal types is a sign of a most extreme compensatory demand made upon the blood-forming tissue, and is only seen in certain very severe conditions. In the child, whose undeveloped tissues are not so far removed from those characteristic of fetal life, reversion of the blood-forming tissues to the fetal type takes place more easily, although still only under conditions of comparative severity. The younger the child, the more readily does this take place. For this reason embryonal cells are more likely to be found in early life in anemic conditions of less severity than in adults, and their presence has less significance.

The pathological changes in anemia are not confined to the blood. There are also changes in the blood-forming organs. These changes may be degenerative or compensatory, according to the pathogenesis of the particular etiological form of anemia which is present. Degenerative changes may or may not be recognizable, according to whether the cause interferes only with the function of the blood-forming tissue, or affects its structure. Even when structural changes are present, they are usually no more than an underdeveloped condition, or an atrophic condition due to impaired nutrition. The compensatory changes are more important. They are seen only in conditions where the pathogenesis of the anemia is increased blood destruction. They consist mainly in proliferative changes in the hematopoietic organs, and are seen mainly where myeloid parenchyma is already present, that is, in the bone marrow. Under certain severe conditions, changes are found also in organs which took part in the formation of erythrocytes during fetal life, but which have ceased to have this function. Such organs are the spleen, liver, and lymphnodes. In them the effort to revert under stimulus to the fetal function is manifested in anemia only by proliferative tissue changes, by hyperplasia. In leukemia, such reversion is more complete, and there is actual new formation of myeloid tissue. In early life the tendency to react to the stimulus of anemia is more marked than in adult life, particularly in the spleen, and it is for this reason that hyperplasia of the spleen is so often seen in certain of the anemic conditions of early life.

CLASSIFICATION.—The difficulty encountered in satisfactorily classifying the anemias is obvious from the nature of the disease. The multiform etiology, the fact that several causes may be associated with a given case, and the fact that the etiology of many cases is partly or wholly unknown, all these things reveal the practical impossibility of classification upon an etiological basis. A common clinical classification is the division into (1) *primary* or idiopathic anemia, and (2) *secondary* anemias. Under this classification a primary anemia means a genuine disease of the blood itself, while a secondary anemia means an alteration in the blood due to the injurious effect of disease of some other organ, or to some known etiological factor. Under this division, the Addison-Biermer type, commonly known as *pernicious* anemia, and the type known as *chlorosis*, were classified as primary, while most other anemias were regarded as secondary. The primary or secondary nature under this classification of the particular type in infancy known as *anemia infantum pseudoleukemica*, or Von Jaksch's anemia, has been a subject of controversy.

The value of this classification lies in the fact that it lays stress upon a feature of the greatest practical importance for purposes of prognosis and treatment, this feature being the known or unknown character of the cause. If this feature had been the only basis used in the division, this classification would have been of the greatest practical value. Unfortunately an effort was made to demonstrate a characteristic pathological anatomy of the blood in the anemias of known and unknown cause respectively. This led to a departure from a division based wholly on the known or unknown character of the cause, and to a placing of certain forms of anemia in the primary or secondary groups because the changes in the blood resembled other cases rightly belonging in those groups. The correspondence between blood changes and cause may be fairly close in adults, but breaks down wholly in children. Many cases of anemia of known cause in early life have been classified as pernicious anemia or chlorosis because the blood changes resembled that seen in those types, yet they were cases obviously secondary to a known cause. On the other hand, many cases in infancy have been classified as secondary on a basis of blood examination, in which the cause was wholly unknown.

It is difficult to understand just what is meant by the term idiopathic disease of the blood. Presumably it means that the lesions are confined to the blood only. But the blood is not a tissue or organ in the ordinary sense of those terms, for the red blood corpuscles do not multiply in the blood itself. It is the result of the complex work of many tissues and organs. Therefore, in the ultimate analysis, there can be no such thing as a primary anemia. Every anemia must be secondary to some deleterious influence acting on the blood or on the blood-making organs.

There are various types of blood changes seen in the anemias. In some cases there is a diminution of hemoglobin only, while in others there is a diminution in the number of red corpuscles. In some cases nucleated erythrocytes are present, in others absent. Embryonal cells—megaloblasts, macrocytes, myelocytes,—may be present or absent. In some cases there may be leukocytosis, in others a normal or diminished white count. It is quite possible to differentiate different types of blood seen in anemia, and to classify the anemias upon this basis. If there were a correspondence between the pathological anatomy of the blood, and the etiology and pathogenesis of the anemias, such a classification would be the one most desirable. The lack of correspondence between the blood changes and the etiological factors, which has been described above, is as serious an obstacle to classification upon an anatomical basis as it was to classification upon an etiological basis.

Ehrlich attempted a classification of the anemias upon the basis of the regenerative forms that appear in the blood. His studies showed that in some anemias the predominant regenerative form is normoblastic or postembryonal, while in others it is megaloblastic or embryonal. In posthemorrhagic anemias and in chlorosis no megaloblasts are seen, and if nucleated erythrocytes appear, they are normoblasts. In pernicious anemia and in anemia infantum pseudoleukemica, on the other hand, embryonal types of cells are present. Ehrlich subdivided the anemias into:—

1. Anemias of the type with postembryonal blood formation.
2. Anemias of the type with embryonal blood formation.

The most recent studies suggest that some megaloblasts may appear in almost any form of anemia, except perhaps in chlorosis. The appearance of embryonal types seems to depend upon the age of the patient and the intensity of the stimulus to the hematopoietic organs, rather than upon the nature of the cause. The appearance of embryonal cells is particularly characteristic of the anemias of early life, and is seen not only in anemia infantum pseudoleukemica, a condition of supposedly unknown cause, but also in many severe anemias which are obviously secondary.

The most modern classifications of the anemias, those found in the most recent works on the subject, are based on a combination of what is known about pathogenesis, and of what is known about etiology. The adoption of such a method of classification is largely due to the writings of Morawitz, whose main divisions are (1) anemias due to increased blood destruction, and (2) anemias due to diminished blood formation. An example of such a classification, not exactly Morawitz's, but based on his ideas, is the following:

I. Anemias due to loss or increased destruction of the blood.
    1. Anemias due to hemorrhage.
    2. Anemias due to known chemical toxins.
    3. Anemias due to bacterial or metabolic hemolytic toxins.
    4. Anemias due to the dibothriocephalus latus.
    5. Hemolytic anemias of unknown etiology.

        - a. Acute types with leukocytosis.
        - b. Chronic type ("pernicious anemia.")
        - c. Anemia infantum psuedoleukemica.
- II. Anemias due to defective or diminished blood formation.
    - 1. Congenital aplastic anemia.
    - 2. Anemias due to new growths or sclerosis of the bone marrow.
    - 3. Anemias due to nutritional disturbance.
    - 4. Anemias of unknown etiology.
        - a. Chlorosis.

Such a classification as the above is by no means wholly satisfactory. It is particularly ill adapted to clinical description, on account of the failure of correspondence between the type of anemia and the changes found in the blood. Furthermore, it is by no means consistent. For example, in the anemias accompanying the chronic infections we do not know whether the pathogenesis is increased blood destruction from bacterial hemolytic toxins, or diminished blood formation from the effect of the toxins on the nutrition and function of the bone marrow.

Nevertheless, this classification is highly useful for purposes of reference. It summarizes what is known of the etiology and pathogenesis of most well-known clinical types of anemia, and shows at a glance just where each type belongs.

In describing the anemias, I shall divide them into the following two groups:—

1. Anemias of known or recognizable etiology.
2. Anemias of unknown etiology.

The essence of diagnosis is not the mere giving of a name, but is the recognition of the cause of the pathological condition. It is knowledge of the cause or causes of a disease which above all else enable us to estimate prognosis, and to plan appropriate treatment. The object of clinical description is to enable the reader to recognize the known or unknown nature of etiological factors in the various conditions encountered. The recognition of the existence of anemia is easy. Laboratory methods show at once the pathological changes in the blood. The problem is to find and remove, or treat the cause, if that is possible.

ANEMIAS OF KNOWN CAUSE

SECONDARY ANEMIA

The term secondary is here used, not as applying to conditions with any particular pathology of the blood, but only as a synonym of "with known or recognizable cause."

ETIOLOGY. DEVELOPMENTAL.—The cause here is congenital hyploplasia of the bone marrow. The type has been described by many clinicians under the name of aplastic anemia. The pathogenesis is diminished blood formation.

MYELOPATHIC.—The cases encountered in early life of anemia due to tumor or sclerosis of the bone marrow, are rare. The pathogenesis is diminished blood formation.

MECHANICAL.—Anemia due to *hemorrhage* is comparatively rare in early life. It is seen after *traumatic* hemorrhage, and in the hemorrhagic diseases,—scurvy, purpura, hemophilia, and hemorrhagic disease of the newborn.

NUTRITIONAL.—Nutritional disturbance involving the blood-forming apparatus is the cause of the largest percentage of cases of anemia seen in early life. The commonest cause of disurbance of nutrition in early life is *faulty feeding*. This may act in two ways in producing anemia. In the first place there may be a deficiency of iron in the food. The full supply of iron needed for purposes of hemoglobin formation cannot be obtained from the food given in the first year of life, whether that food be human milk or cow's milk. In neither is there sufficient iron, and the infant must normally depend upon a surplus of iron stored in the liver during fetal life. This is usually sufficient for the first year, but exclusive milk feeding after the first year is a common cause of the development of anemia. Even if the diet in the second and third years is not exclusively milk, a badly balanced diet with an insufficiency of iron-containing food will lead to the development of anemia. In some infants this purely *dietetic* anemia develops prematurely during the first year, the cause being an insufficient amount of the stored iron surplus. Such cases are not uncommon in infancy, and the possibility of this cause must be remembered before anemia in an infant is designated as primary.

Another presumably nutritional cause of anemia in infancy is chronic functional gastro-intestinal disturbance. The pathogenesis of these cases is, perhaps, not fully understood. The disturbance resulting in anemia cannot be due to mere lack of food material, because anemia is not a marked feature in experimental starvation, the atrophy of the blood appearing to keep pace with that of the rest of the body. Also, considering the great frequency of chronic gastro-intestinal disease in infancy, anemia is found as an associated condition much less frequently than one would expect. The majority of the cases of "infantile atrophy" are not anemic. There are cases, however, in which anemia comes on with gastro-intestinal disorders, and disappears when the nutrition improves. In such cases we cannot know whether the anemia is produced by some disturbance of metabolism which particularly affects the blood-forming apparatus, or whether the nutritional injury comes from external factors in hygiene and environment which are also a contributing cause to the gastro-intestinal insufficiency.

Certain specific disorders of metabolism and nutrition are usually associated with anemia. The most important is the disease *rachitis*. Here again, we cannot know whether the anemia is produced by the external factors of hygiene and environment which are known to be etiological factors in rickets, or by some more specific effect. These hygienic factors, we know, can produce anemia without rickets. On the other hand, in some cases of rickets, anemia of a particularly severe type is seen, in which the changes in the blood suggest a pathogenesis of increased blood destruction rather than of diminished blood production, and such cases are strongly suggestive of a definite connection between the disturbance of metabolism present in rickets and the pathological changes in the blood.

Finally, there is a large group of anemias seen mainly in older children, but also in infants, in which the cause is undoubtedly *faulty hygienic surroundings*. These lead to a disturbance of nutrition which affects the function of the bone marrow in such a way as to cause diminished formation of hemoglobin and erythrocytes. The factors are crowding, unhealthy dwellings, insufficient air, insufficient sunlight. The rapid response of these cases to no other treatment than change of environment is sufficient proof of their cause.

TOXIC.—Anemia from exogenous chemical toxins is so rare in early life, that it need only be mentioned for the sake of completeness. The pathogenesis is increased blood destruction.

The effect of toxins of endogenous origin, exercised either upon blood formation or upon blood destruction, cannot be excluded in an anemia due to any diseased condition associated with disturbance of metabolism. The severe anemia seen in general sarcomatosis suggests a toxic origin. The chief condition in which the marked accompanyng anemia is certainly due to the effect of endogenous toxins, is the disease *nephritis*. The pathogenesis is not known.

The largest group of anemias of toxic origin includes those due to bacterial toxins. The severe anemias often associated with the chronic infections, *tuberculosis* and *syphilis*, are probably the result of a chronic intoxication which injures both the blood and the blood-forming mechanism, although they might be explained by the nutritional disturbance which accompanies these chronic infections. That anemia may certainly be due to the effect of bacterial toxins is evident from the rapid development of the condition in certain of the acute specific infections, notably in *rheumatic fever*, and in *diphtheria*. In *malaria*, the severe anemia is due to demonstrable destruction of red corpuscles. The severe anemia which often accompanies certain *suppurative processes*, such as empyema, is well known. In all these anemias of probable toxic origin, Morawitz's sharp differentiation into types with the pathogenesis of increased blood destruction, and types with the pathogenesis of diminished blood formation, cannot be made. It is probable that the toxins have both effects, with a predominance sometimes of one, sometimes of the other.

The anemias associated with certain of the animal parasites also belong in the toxic group. The most notable form is that produced by the dibothriocephalus latus, in which the blood presents the picture formerly supposed to be characteristic of primary pernicious anemia. The pathogenesis of these anemias is evidently increased destruction.

SYMPTOMS.—Anemia in early life has no subjective symptoms peculiar to the condition. Any symptoms which may be present are those of the disease to which the anemia is secondary. The condition of anemia is to be suspected when physical examination shows pallor of the skin and visible mucous membranes. Its presence is confirmed by the examination of the blood. The only important symptoms are the changes always found in the blood, and sometimes found in the spleen.

BLOOD CHANGES.—The changes in the blood in the anemias of known cause encountered in early life vary chiefly with the severity of the anemia. They also vary to some extent with the nature as well as with the severity of the etiological factors, but in my opinion this variation is not sufficiently constant to permit the description of a characteristic blood picture for each etiological group.

The mildest cases may show a moderate diminution in the percentage of hemoglobin only, or a moderate diminution in both hemoglobin and red blood corpuscles. It is probable that in cases in which the anemia is due to decreased blood formation, the hemoglobin suffers first, and in the early stages of such cases the blood presents the characteristics formerly associated only with a mild type of chlorosis. Later, or in more marked cases of this pathogenesis, there is diminution in the number of red corpuscles as well. In cases in which the anemia is due to increased blood destruction, the red corpuscles are diminished from the start. In all these mild types, there are no other notable blood changes, the diagnosis resting simply on the decreased hemoglobin percentage and red cell count.

In more severe cases, there are abnormalities in the size, shape, and staining reaction of the red corpuscles, and nucleated red corpuscles appear. The erythroblasts are usually mainly of the normoblastic type, but in young infants megaloblasts are often seen. Many writers agree with Morawitz in believing that the appearance of erythroblasts is characteristic of anemias due to increased blood destruction, and that these cells are few or absent in anemias due to diminished blood formation. I do not think that this rule is very constant, especially in the anemias of early life. The theory on which it is based is that the appearance of erythroblasts is an evidence of regenerative activity in the bone marrow, which only takes place in normal tissue under the stimulus of increased blood destruction. Nevertheless the analogy of other tissues suggests that regenerative activity in a tissue can and usually does take place after injury to the tissue, all parts of the tissue never being equally injured. Even in posthemorrhagic anemia erythroblasts are sometimes few or absent, while on the other hand, in early life at least, erythroblasts are often numerous in cases of probable nutritional origin. Nevertheless, the rule may be of some aid in the search for the cause of the condition.

Abnormalities of the red cells--variations in size, poikilocytosis, polychromatophilia, and the presence of erythroblasts—are particularly characteristic of the anemias of infancy. In older children they are a sign of a very severe degree of anemia. In infancy they are likely to appear in anemias of comparatively less severity. This is to be explained by the fact that in infancy the hematopoietic tissue is already working to the limit of its capacity, and being unable to increase its productive power by hyperplasia, it compensates for the anemia by the hurried production of imperfectly formed cells. In cases of extreme severity in older children, normoblasts appear, while in infancy there is often a reversion to a fetal type, with the appearance of macrocytes and megaloblasts.

Leukocytosis is likely to be present in the anemias of infancy in all but the mildest forms. The amount of leukocytosis varies with the severity of the anemia. The type of the leukocytosis varies somewhat with the cause, depending on the chemotactic influence of the various etiological factors, but here again the correspondence between blood picture and cause is not very constant, there being many unknown factors. The most common type is polymorphonuclear, but a relative lymphocytosis is not uncommon, especially in the younger infants, and in cases due to gastro-intestinal disease. In malaria there is a relative increase in the large mononuclear basophiles, while in anemias caused by the animal parasites, as in helminthiasis, the leukocytosis is eosinophilic. In the severer forms in infancy *myelocytes* are frequently found. Their number is never so great as to suggest myelogenous leukemia, but may reach ten per cent or more.

The polymorphism of the blood in severe cases of anemia in infancy associated with marked hyperplasia of the spleen, led to the differentiation of a special type of anemia, called *anemia infantum pseudoleukemica,* or von Jaksch's anemia. Discussion has raged around the question whether such cases represent a secondary anemia or a primary disease of the blood. The separate identity of the condition as a disease of the blood cannot rest upon pathological anatomy, as neither the changes in the blood nor in the spleen present anything specific. The changes in the blood are merely those of a severe anemia modified by the peculiar characteristics of such conditions in infancy. The changes in the spleen are merely those of a chronic hyperplasia. The separate identity of anemia infantum pseudoleukemica can only be based on a specific etiology. In a certain number of cases presenting the clinical picture of von Jaksch's anemia,—diminished red count, erythroblasts including many megaloblasts, myelocytes, increased eosinophiles, and marked splenic enlargement—a competent etiological factor, such as rickets, has been found. In the majority of cases, no cause has been found, and these should be placed among the anemias of unknown cause. In Italy, and along the shores of the Mediterranean, a certain number of cases presenting the clinical picture of von Jaksch's anemia have been shown to be caused by a parasite, which is found in the spleen, and which closely resembles the Leischman's parasite of the tropical disease Kala Azar. These cases run a febrile course, and were originally described by Italian writers as the Infectious Anemia of Infants.

Most foreign writers have described a severe type of anemia characterized by marked diminution both in the hemoglobin and in the red corpuscles, but without the presence of any cells of the regenerative type, or of splenic enlargement. This condition is described as *aplastic anemia*, and there is evidence that it is due to hypoplasia of the bone marrow. It probably accounts for certain severe cases developing in early infancy without obvious cause.

THE SPLEEN.—The spleen is frequently enlarged in the anemias of infancy, less often in later childhood. The degree of enlargement varies both with the severity of the anemia, and with the nature of the cause. Only slight or no enlargement is seen in some severe anemias, while in others the organ is enormously increased in size. The pathogenesis of the splenic enlargement has been described above. Enlargement of the spleen to a greater or less degree is almost constant in the anemia accompanying rickets, but as it is also seen in rickets without marked anemia, it is difficult to tell whether the hyperplasia is secondary to the anemia, or to the rickets.

DIAGNOSIS.—The diagnosis of anemia from the blood examination is simple enough. The important point to be determined is the nature of the cause. In later childhood the examination of the blood is of some help in determining the character of the etiology. The types of blood associated with chlorosis and the Addison-Biermer type (pernicious anemia), the two principal forms with unknown etiology, are distinctive. Nevertheless both are rare in childhood, and the presence of a characteristic blood picture should not preclude a careful search for all possible causes. Many cases of mild anemia in childhood due to faulty hygienic surroundings present a blood picture resembling that of the true chlorosis of unknown etiology seen so often a little later in life. In general, unless the blood shows definitely the characteristics of the "pernicious" type, anemia in older children can safely be diagnosed as secondary. The cause can usually be found, either in some recognizable disease, or in faulty conditions of hygiene and environment.

In infancy the finding of the cause is more difficult. The blood picture is by no means so helpful in enabling us to place the case in the group with known or in the group with unknown etiology. In the milder anemias the blood often resembles that of chlorosis, although an adequate cause may be present. If the term chlorosis be limited to a condition of constantly unknown etiology, rather than applied to a certain blood picture, it probably can never be properly applied to a case of anemia in infancy. In other cases in infants the blood picture often bears more or less resemblance to that of the pernicious type. As typical cases of this type with unknown etiology are almost unknown in infancy, such a blood picture should suggest a toxic etiology, and every effort should be made to discover an adequate cause.

In the severe anemias with marked splenic enlargement, the determination of the etiology of the condition is often still more difficult. When the blood picture resembles that usually associated with "secondary anemia," the history and physical examination of the patient will often reveal an obvious cause for the anemia. When on the other hand the blood picture reveals the polymorphism characteristic of the severe anemias of infancy, an adequate cause may often be difficult to find. The cases of anemia with marked splenic enlargement and with a blood picture resembling that usually described as characteristic of anemia infantum pseudoleukemica, are particularly difficult to classify etiologically. Even when some possible cause such as rickets or prolonged gastro-intestinal disturbance is present, the physician may be in doubt as to whether the severe anemia with splenic enlargement is wholly due to this cause, or whether there is some potent unknown factor. The "secondary" character of these cases, the term secondary meaning "with known cause," must always be a matter of some doubt.

The purely "alimentary" type of anemia, due to a relative insufficiency of iron in the diet, is easier to recognize. Usually there is a history of too prolonged milk feeding, or of some unsuitable method of feeding. The blood shows a picture which in the earlier stages resembles that of chlorosis, and which later shows diminution in the number of red cells without the presence of splenic enlargement, of erythroblasts, or of cells showing irregularities of size, shape, and staining reaction.

The "aplastic" type is to be suspected when severe anemia begins early in infancy, without the history or physical examination revealing any adequate cause. Splenic enlargement and regenerative cells in the blood are both absent.

In general the diagnosis of the etiologic form of secondary anemia must depend mainly on the physician's knowledge of the etiological factors concerned in anemia. It depends more upon the careful search for possible causes, as carried out by a careful history and physical examination, and by the use of all the available special and laboratory methods of clinical examination, than upon the examination of the blood alone.

PROGNOSIS.—The prognosis depends mainly on the nature of the cause. The anemias seen in later childhood are almost never serious, the cause being usually factors in hygiene and environment which are not only easily discoverable, but usually easily removable. In anemias due to definite diseases, such as tuberculosis, syphilis, rheumatic fever, prolonged suppuration, nephritis, diphtheria, malaria and so forth, the prognosis is that of the disease, the anemia being only a symptom. In the anemias due to accidental traumatic hemorrhage, the blood is rapidly restored. In anemias due to the hemorrhagic diseases, such as scurvy and purpura the prognosis is that of the underlying disease.

In the anemias of infancy, the prognosis is more doubtful, because the etiology is more uncertain. In the milder anemias associated with poor hygienic surroundings, faulty feeding, gastro-intestinal disturbance, and rickets, the outlook for improvement with removal or proper treatment of the cause is excellent. In the severe anemias with splenic enlargement, the prognosis is much more uncertain. In the first place there is the ever-existing doubt as to whether the possible etiological factors which are present represent the whole of the etiology, or whether there are other unknown factors of a more serious nature which can not be removed. Again, in all the diseased conditions of infancy, a marked degree of anemia, even if due to a cause which is eventually removable, may be a contributing factor of sufficient severity to turn the scale against the baby before the treatment of the underlying condition has had time to take effect.

In the rare aplastic type of anemia, the prognosis is rather unfavorable, the patient falling an easy victim to the various common diseases of early life. In the alimentary type, the prognosis is good if proper treatment be instituted before the child's vitality and resistance are markedly lowered.

TREATMENT.—In all cases of anemia belonging to this group, the principal essential therapeutic indication is removal of the cause, or treatment of the cause. The most important part of the treatment in the majority of cases of secondary anemia is hygienic and dietetic. The feeding of the child and every detail of the child's environment should be carefully scrutinized, and should be regulated in accordance with the principles described in the first part of this book as being those essential to the maintenance of normal development.

In the anemias due to faulty surroundings, absence of exercise, fresh air and sunlight, and so forth, the bettering of these conditions will prove to be a specific measure in treatment. In anemias due to prolonged or exclusive milk feeding, or to any other fault in diet, the regulation of the feeding with a mixed diet containing fruits, beef-juice, eggs and green vegetables will effect a removal of the cause of the anemia. In anemias secondary to gastro-intestinal disease, dietetic treatment of the underlying condition will prove most effective in overcoming the anemia. In the anemias secondary to other recognized diseases such as the infections, rickets, scurvy, purpura, nephritis and so forth, the treatment of the anemia is that of the underlying disease. The general hygienic and dietetic treatment of anemia is of far more importance than the administration of drugs. Nevertheless, drug treatment should not be omitted in these cases.

The drugs which are most useful in the treatment of anemia are *iron* and *arsenic*. Iron is the most useful in the treatment of anemias which are secondary to known causes. It is obviously indicated in the cases of anemia in infants which are due to prolonged milk feeding, or to an improperly balanced diet. In such cases the supply of iron stored in the liver is insufficient to meet the demand for iron to be used in making hemoglobin, and this deficiency can be supplied by the giving of an iron preparation. Furthermore, in any of the anemic conditions of infancy, an insufficient supply of stored iron may be a contributing factor. Not only, however, is iron of benefit in making up for a deficiency, but there is evidence that it also acts as a stimulus to the hematopoietic tissues. The value of arsenic in anemia depends upon the same stimulant action.

A great many preparations of iron are available for use in infancy and childhood. In the anemias of infancy which are so frequently caused or complicated by gastro-intestinal disturbance, the giving of iron by mouth may disturb digestion, and furthermore, in such conditions the absorption of iron is rather uncertain. For this reason I am accustomed to give iron subcutaneously in all the severer anemias of infancy. The best preparation is the *citrate of iron*, which comes in glass ampules prepared for subcutaneous injection. The dose is three-quarters of a grain given every other day or every third day. In the milder cases of anemia in the first two or three years of life, the best preparation is the *saccharated carbonate*, given in doses of from 3 to 5 grains three times a day. In older children who are able to swallow pills, I prefer *Blaud's pills* rather than ovoferrin or other synthetic preparations of iron. Other preparations adapted to use in early life are the albuminate, the bitter wine, the sweet wine and the malate, given in doses proportioned to the age of the child. The giving of arsenic in combination with iron, or at times in alternation with iron, is recommended by most authorities as being useful. The evidence of benefit is of the uncertain clinical variety, but there is no reason why this method of treatment should not be tried. It is quite possible that there are cases in which arsenic meets better the requirements of the etiological factors.

Very severe or resistant cases of anemia are sometimes encountered, especially in infancy, in which the anemia becomes a condition which actually threatens the life of the child. In such cases *transfusion* is indicated. This measure will often produce a temporary benefit of sufficient duration to allow treatment of the underlying cause to take effect.

ANEMIAS WITH UNKNOWN CAUSE

PRIMARY ANEMIA

Under this heading the term primary is taken as synonymous with " of unknown cause." The cases which come into this group can obviously not be classified upon an etiological basis. We can only classify them upon a symptomatic basis, that is, upon the basis of the blood changes. As we have seen, these changes are in no way characteristic of the anemias in this group, the same changes being seen in the secondary anemias. In the anemias of unknown cause there are three main clinical types. These are known as chlorosis, pernicious anemia, and anemia infantum pseudoleukemica.

CHLOROSIS

If the term chlorosis be understood to apply only to a condition of anemia of unknown cause and not to all cases in which the blood shows certain characteristics, it is doubtful whether the disease ever occurs in infancy. It is also extremely rare throughout the greater part of childhood. It is seen occasionally in the last years of childhood, and is most common in young girls at about the time of puberty.

ETIOLOGY.—Very little is known about the cause of this type of anemia. Various factors have been found associated with certain cases, such as heredity, unhealthy occupations, unhygienic surroundings, insufficient food or clothing, psychical disturbance, mental or physical over-exertion and disorders of menstruation. But none of these factors are associated with the condition with sufficient frequency or constancy to be considered as actual etiological factors. The disease is rare in males, being usually seen in girls between the thirteenth and seventeenth years of life, more often in blondes than in brunettes.

PATHOLOGICAL ANATOMY.—Chlorosis is rarely fatal. In some cases hypoplasia of the heart, large vessels, ovaries, and uterus has been found, also considerable fatty degeneration of the heart and of the intima of the vessels. Gastric ulcer has been occasionally associated with it.

SYMPTOMS.—Subjective symptoms are commoner in chlorosis than in the anemias of earlier childhood. The patient often complains of shortness of breath on exertion, and attacks of palpitation and syncope are common. The appetite is poor or perverted, and there is a craving for unusual and indigestible articles of diet. Gastric hyperacidity is often present, and almost always constipation. Enteroptosis is not uncommon. The patient frequently complains of headache or neuralgic pains. Hysterical manifestations may occur. The menstrual function is often disturbed; the flow is scanty, irregular, and sometimes painful. The patient is usually plump and soft, and the skin has a peculiar greenish-yellow tint which gives the disease its popular name of " green sickness." Systolic murmurs are frequently heard over the heart in the pulmonary or mitral region, and also a venous hum, the " bruit de diable," in the veins of the neck. Palpitation is occasionally seen in the jugular veins. There is occasionally a tendency to venous thrombosis, which most often occurs in the femoral veins. The pulse is full and soft. Rarely there is some puffiness of the face, and edema of the ankles. The urine rarely contains a slight trace of albumin. Gastric ulcer sometimes occurs as an associated condition.

THE BLOOD.—The essential feature of the blood changes in chlorosis is diminution in the hemoglobin, which is very marked in proportion to the reduction in the number of erythrocytes. The hemoglobin is frequently as low as 30 or 40 per cent. The red cells are usually from 3,500,000 to 4,000,000 per cubic millimeter, and are rarely lower than 3,000,000. The specific gravity of the blood is reduced in proportion to the loss of hemoglobin, usually not below 1.035. Morphologically the red corpuscles appear pale with a wide clear area in the center. There is very little if any irregularity in size and shape. Normoblasts are very rarely found, and megaloblasts are probably never present. The number of leukocytes and the relative number of the various forms of leukocytes are usually unchanged, although in some cases a moderate relative lymphocytosis has been reported.

DIAGNOSIS.—The age and sex of the patient, the well-nourished condition, the pearly sclerotics, the color of the skin, and the character of the blood form a striking clinical picture which easily distinguishes chlorosis from other forms of anemia.

PROGNOSIS.—The prognosis is almost always good. The disease often lasts a year and relapses are common. Chlorosis is rarely fatal, except when complicated by tuberculosis or gastric ulcer.

TREATMENT.—There are few therapeutic measures in medicine more satisfactory than the use of iron in chlorosis. The best preparation is Blaud's pills. It is usually necessary also to treat the constipation which is so frequently present. Appropriate doses of nux vomica are often useful. Arsenic is often used in addition to the iron. The removal of all unfavorable dietetic and hygienic factors is always indicated. A change of scene and the removal of the patient from sources of nervous excitement or disturbance, are often very valuable.

PERNICIOUS ANEMIA

This is the most severe type of the primary anemias. It is characterized by very uniform changes in the blood and by a progressive course, which leads in the majority of cases to a fatal termination. It is a very rare disease in children and is almost unknown in infancy. Many of the reported cases in infancy have not been true cases of pernicious anemia, but have been severe anemias belonging in the group of anemia with known cause which were diagnosed as pernicious simply on the basis of the changes in the blood.

ETIOLOGY.—The cause of the disease is entirely unknown. The constancy of the clinical picture and of the course of the disease is highly suggestive of some specific cause, but no associated conditions have been found with sufficient frequency to enable us to regard them as possible etiological factors. The changes in the blood and the reaction of the hematopoietic tissues strongly suggest that the pathogenesis of the disease is a toxic hemolysis. There is no doubt that pernicious anemia belongs in the group with increased destruction of erythrocytes, and with marked compensatory efforts in the hematopoietic tissues. The origin and nature of the toxins however are as yet undiscovered.

PATHOLOGICAL ANATOMY.—The body is not often emaciated. The skin usually shows a lemon-yellow tint. There is marked anemia of the organs at times, with many capillary hemorrhages into the organs and skin. The heart is large and flabby, and shows intense fatty degeneration. The liver is fatty and sometimes enlarged. The kidneys and blood vessels show fatty changes. One of the most characteristic features is a deposit of iron in the liver tissue and often in the intestinal mucosa. The spleen is hard and somewhat increased in size as a result of overgrowth of the fibrous tissue of the pulp. Some of the lymphnodes are often enlarged. The bone marrow is " splenified," that is, returned to its embryonic state, with a predominance of megaloblasts.

SYMPTOMS.—The general symptoms are those of severe anemia, increasing pallor and prostration, usually without emaciation. The onset is insidious, and the clinical course does not differ from that of adults. There is frequently more or less gastro-enteric disturbance or dyspnea. Hemorrhages occur into the skin or from the nasal and other mucous membranes. Edema often appears in the dependent portions of the skin, less frequently in the cavities of the body. Hemic murmurs are usually heard in the cardiac region. The pulse is full and soft. It is to be noted that hemic murmurs are less frequent in the anemia of infants than in that of adults. The urine is scanty and of low specific gravity. The temperature is variable; at times it may be normal, and at others an irregular pyrexia may run for a considerable period. The course is a chronic one, but varies in length. The disease may be completely developed in some cases in two or three months, and in others only after ten or twelve months. In most instances it progresses steadily to a fatal termination.

THE BLOOD.—The red corpuscles are much diminished, usually to one million, sometimes even to five hundred thousand per cubic millimeter. The hemoglobin is diminished per bulk of the blood, often to 25 or 30 per cent, rarely to 12 or 15 per cent, but owing to the greater reduction of red cells it is often relatively increased per corpuscle (the color index is high and sometimes greater than normal). This is exactly the opposite condition to that which occurs in chlorosis. The red cells show great variation in shape and size, many oval, rod-shaped, or irregular corpuscles being present, which take stains in an uneven way. A majority of the red cells are larger than normal (macrocytes). Many nucleated red cells are present, both of normal size (normoblasts) and of larger size (megaloblasts). The megaloblasts are the more numerous of the two. Many of the nuclei are found in a process of division (karyokinesis). The leukocytes are normal in number, or somewhat diminished,—8,000 to 4,000. In the more severe cases there is frequently a relative increase of the lymphocytes with a corresponding diminution of the polynuclear cells. A few myelocytes are not infrequently found.

DIAGNOSIS.—The blood picture is so characteristic that it is unmistakable. The most characteristic features are the great diminution in the number of red cells with a high color index, the predominance of megaloblasts over normoblasts, and the normal or diminished leukocyte count. In adults such a blood picture is particularly diagnostic of pernicious anemia, the only other condition showing such changes being the rare cases of anemia due to the dibothriocephalus latus. In early life, more especially in infancy, the blood changes alone are not a sufficient basis for the diagnosis of primary pernicious anemia. The reaction of the blood of infants to some of the known causes of severe anemia is so variable that occasionally a clinical picture much resembling that of pernicious anemia is produced. Therefore, it is essential in diagnosis not only that the blood picture be characteristic, but also that no adequate cause for a severe secondary anemia can be found.

PROGNOSIS.—The prognosis in true cases of pernicious anemia with unknown cause is fatal. The course of the disease is generally more rapid in children than in adults, the duration in most cases being not over six months; as in adults, the course of the disease is marked by periods of exacerbation and remission, but in general the tendency is progressively downward to a fatal ending.

TREATMENT.—Treatment is undertaken rather with a view to the possibility of a mistake in diagnosis, than with any hope of effecting a cure if the diagnosis be correct. Arsenic is more useful than iron in this form of anemia. It is usually well borne, and should be given in small doses, gradually increased to the limit of tolerance. A child of ten years can usually be worked up to take 30 minims of Fowler's solution in the twenty-four hours. Arsenic may also be given in other forms. Active exercise should be prohibited and replaced by massage, which is of great value and never to be neglected. Rest in bed, either absolute or for the greater part of the time, is essential. Small, frequent and regular meals should be given, of light, nutritious food. Bone-marrow in the form of a glycerin extract has been recommended, but has thus far proved of doubtful value. The free administration of oxygen day and night for several weeks is worthy of trial, if expense is not to be considered.

ANEMIA INFANTUM PSEUDOLEUKEMICA

(Von Jaksch's Anemia; Anaemia Splenica Infettiva dei Bambini;
Splēnomegalie Primitive; Splenic Anemia of Infancy)

This form of anemia was first described by von Jaksch in 1889, and was believed by him to be a special disease entity peculiar to infants and young children. The discussion which has raged around the separate identity of this disease has already been referred to.

If, however, the term anemia infantum pseudoleukemica be confined to cases of anemia of unknown etiology with full recognition of the fact that exactly the same disease picture can be produced by many known causes, the question of the separate identity of the disease loses its interest and importance. Under this limitation the disease represents a severe anemia with splenic enlargement of unknown etiology, seen only in infancy and early childhood. The term pseudoleukemica is a misnomer. It was originally adopted on the ground that von Jaksch's anemia is an infantile form of Hodgkin's disease (pseudoleukemia). It has since been shown that the changes in the spleen are entirely different from those of Hodgkin's disease, which is now a condition of known etiology. The changes in the spleen are similar to those found in Banti's disease, which is a primary disease of that organ. Nevertheless, the weight of evidence suggests that in anemia infantum pseudoleukemica the primary changes are in the blood, and the hyperplasia of the spleen is secondary to the anemia. For this reason the disease is to be differentiated from Banti's disease, and the synonym splénomegalie primitive is not applicable.

ETIOLOGY.—The cause of the disease is entirely unknown. The process seems to be a severe anemia of the pathogenetic type of increased blood destruction with compensatory changes most marked in the spleen. In a certain number of cases there are evidences of rickets, and such cases are difficult to classify upon an etiological basis, the question arising as to whether the actual cause of the disease is the rickets, or some unknown factor. The majority of cases occur in infants between the ages of seven and twelve months.

PATHOLOGICAL ANATOMY.—The most constant lesion is that of the spleen, which is very markedly enlarged. It is firm and hard, and there may be evidences of a perisplenitis. The microscope shows that the changes are those of simple hyperplasia. Enlargement of the liver is seen in some cases, but is much less marked and less constant than in Banti's disease, there being no relation between size of the spleen and that of the liver. The hepatic cells are unchanged. Some enlargement of the lymphnodes is seen in about half the cases. It is rarely marked, and the changes are those of simple hyperplasia. Changes in the bone-marrow are inconstant, and when present are suggestive only of compensatory hypertrophy.

SYMPTOMS.—The onset of the disease is gradual. The nutrition is poor; there is considerable emaciation, a waxy tint of the skin, at times hemorrhages from the mucous membranes and into the skin, a much enlarged spleen, and at times a moderately enlarged liver. There is no tenderness over the bones, and there is rarely a venous bruit in the neck or functional cardiac murmurs.

THE BLOOD.—The blood shows the characteristics of an infantile anemia of varying but rather marked intensity, that is, a diminished count of erythrocytes, which may be as low as 1,000,000, but usually range between 2,000,000 and 3,000,000 low hemoglobin, but not always in proportion to the reduction in the erythrocytes, variations in the size and shape of the erythrocytes and in the number of erythroblasts, which are usually numerous and consist mostly of normoblasts. There is no definite relationship between the degree of anemia and the size of the spleen or of the liver. The leukocytes are not characteristic, being markedly increased in most cases and in normal proportions in some. They vary in number from normal to 50,000 per cubic millimeter. In the majority of cases, the larger the spleen the greater is the leukocytosis, according to Hutchinson. Morse dissents from this view. He admits that the largest spleens usually occur in connection with the greatest leukocytosis, but in his series he found occasionally large spleens with no leukocytosis. Myelocytes are usually present in small percentages, but not in such numbers as to suggest a myelogenous leukemia. The lymphocytosis reported by many writers may occur under any condition giving rise to an increased number of white cells.

DIAGNOSIS.—In making a diagnosis we should limit the term anemia infantum pseudoleukemica to those cases in which no adequate cause for secondary anemia can be found, and in which the clinical symptoms and changes in the blood already described are present. The differential diagnosis should first be made from leukemia and Hodgkin's disease. The proportionately low leukocytosis and the absence of either a general lymphatic enlargement or an excess of myelocytes in the blood would differentiate it from the former, while the absence of markedly enlarged lymphnodes would exclude Hodgkin's. As there are so few cases in which a thorough and reliable examination of the organs has been made in which rachitis was not present, the diagnosis between rachitis with enlarged spleen and secondary anemia, and this supposed especial group of symptoms, would be impossible. Banti's disease is differentiated from this form of anemia on the following grounds:—It is not definitely and constantly associated with severe anemia, and does not usually show the changes in the blood characteristic of von Jaksch's anemia; it occurs more often in older children; it is constantly associated with enlargement of the liver, and later with ascites.

PROGNOSIS.—There is much less evidence that anemia infantum pseudoleukemica is associated with a single or specific cause than there is in the case of pernicious anemia. The cause is unknown, but it is quite probable that the same clinical picture may be produced by various causes. While the prognosis is more unfavorable than in anemias presenting a similar clinical picture but with known cause, it is nevertheless by no means so absolutely unfavorable as is that of pernicious anemia. A certain number of cases eventually recover, although many die. Death, however, when it occurs, is by no means definitely to be attributed to the anemia. Most of these infants die from intercurrent disease, to which they appear to be peculiarly susceptible.

TREATMENT.—The general hygienic and dietetic treatment of anemia infantum pseudoleukemica is the same as that of the severe anemias with known causes. With infants who are placed under the best possible conditions of environment, it is especially important that fresh air treatment similar to that used in rickets should be thoroughly carried out. The feeding should be carefully regulated in accordance with the digestive demands of the individual child. In the drug treatment, arsenic is believed by most authorities to offer a better prospect of improvement than iron. It is given in increasing doses as described for pernicious anemia. Subcutaneous injections of citrate of iron may be used in addition.

Some authorities have advocated splenectomy in anemia infantum pseudoleukemica. The basis is the good results obtained by this procedure in Banti's disease. It is now, however, fairly recognized that Banti's disease represents a primary disease of the spleen, and that in the anemias of infants with enlarged spleens, the primary condition is the anemia. Consequently, splenectomy would not be indicated, and in view of the very uncertain prognosis of von Jaksch's anemia, I do not think that splenectomy should ever be considered in cases of this type.

PROBLEMS AND RESEARCH.—The central problem in the anemias is that of etiology and classification. Much progress has been made especially in the recognition of the pathogenesis of the changes in the blood and hematopoietic organs, the recognition of the factors of diminished blood production and increased blood destruction, the recognition of the compensatory changes in the hematopoietic organs, and the recognition of the fact that certain changes in the blood and blood-making organs represent a reversion to a fetal type.

All this is the result of recent work on the anemias and is of the highest value. The unknown facts have to do with the real causes of many forms of anemia, and of the relation of these causes to the changes found in the blood. The literature of the next ten years will undoubtedly shed further light upon these problems.

Much of the literature of the anemias has been concerned with the discussion many times referred to above as to the separate identity of von Jaksch's anemia. In the broader view of the anemias presented here, this question is of little importance. The important question as always, is that of ultimate etiology. Whenever a correspondence between pathological anatomy and symptoms and etiology can be found, it is of value, but in the anemias it is well that we recognize now that such a correspondence never will be found.

A great bulk of European literature has been devoted to the rôle of the Leischmann parasite in cases of the anemia of the von Jaksch's type. The final result of all this discussion has been the establishment of the fact that along the shores of the Mediterranean there are cases with severe anemia and splenic enlargement in which a parasite resembling that of Leischmann can be demonstrated in the spleen. Italian writers therefore classify the splenic anemias of infants in two groups, (1) that with fever due to the parasite, and (2) those without constant fever due to unknown or other causes. As far as I know, the parasite has not been found in this country.

LEUKEMIA

This is a disease in which the essential feature is the extensive proliferation of the leukopoietic tissues, either lymphadenoid or myeloid, with the appearance in the blood of leukocytes not normally present, and usually, but not always, with a great increase in the number of leukocytes.

Leukemia is relatively a rare disease. It is commonest in middle life, but is seen at all ages, even in infancy. It is much less common in children than in adults. This statement, however, refers to the chronic forms, which are the commonest. The acute forms, especially the acute lymphatic, are commoner in children than in adults. Even in childhood, males are affected more frequently than females.

ETIOLOGY.—The cause of leukemia is entirely unknown. Some cases have been reported suggesting an hereditary factor; other cases have followed syphilis, rickets, malaria, and other infections. In the majority of cases it apparently occurs as a primary disease in children previously healthy. Many writers believe that the theory that the disease is of infectious origin is the most probable. Various supposed parasites have been described in the white cells, but there is no evidence that they are etiological agents. It is possible that the disease represents a specific tissue reaction to various obscure toxic agencies, among which infection may hold an important place.

PATHOLOGICAL ANATOMY.—The principal lesions of leukemia are found in the lymphnodes, spleen and bone-marrow. The leukemias are divided into two forms, the *lymphadenoid* and the *myeloid*, according to whether the lesions involve mainly the lymphnodes on the one hand, or the spleen and bone-marrow on the other.

LYMPHADENOID FORM.—In this form, more commonly but less properly called *lymphatic leukemia,* there is a more or less general involvement of the lymphadenoid tissue throughout the body, including the peripheral, tracheobronchial, and mesenteric lymphnodes, the tonsils, the lymphadenoid tissue of the tongue and pharynx, and the Peyer's patches and solitary follicles of the intestine. The parenchymatous organs, particularly the liver, are also involved, the proliferation of lymphadenoid tissue giving rise to the so-called leukemic infiltration. The lesion is a simple hyperplasia. In the spleen and bone-marrow the changes are not proliferation of the splenic and myeloid tissue, but the parenchyma is more or less suppressed by accumulations of lymphoid cells (lymphadenoid tissue).

MYELOID FORM.—In this form, more commonly called *splenomyelogenous* or *splenic myelogenous leukemia,* the principal lesions are in the spleen and bone-marrow. The essential feature is proliferation of the myeloid tissue. In the bone-marrow the proliferation gives rise to extensive accumulations of myelocytes. In the enormously enlarged spleen there is hyperplasia of the trabeculae, and extensive deposits of cells which form scattered light gray areas. These microscopically resemble lymphadenoid tissue, but their perivascular distribution and the finer staining reactions of the cells show that they differ from the lymphadenoid proliferations seen in the lymphatic form of leukemia, and are of myeloblastic origin. Similar perivascular myeloid proliferations are found in other parts of the body, chiefly in the liver, where the leukemic infiltration is usually pronounced, but also at times in the kidneys, in other parenchymatous organs, in some of the lymphnodes, and occasionally in the skin and serous membranes.

For purposes of clinical description the leukemias may be divided into four forms: (1) Acute lymphadenoid; (2) chronic lymphadenoid; (3) acute myeloid; (4) chronic myeloid.

ACUTE LYMPHADENOID LEUKEMIA

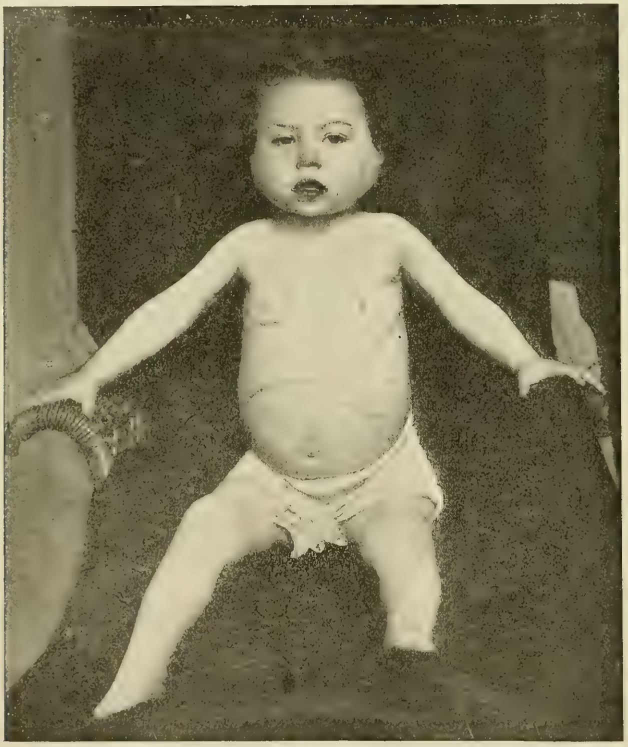
(Acute Lymphatic Leukemia; Acute Leukemic Lymphadenosis)

This is the only form in children which is not a great rarity.

SYMPTOMS.—The onset is sudden, and resembles that of an acute infectious disease. There is a rapid rise of temperature with headache and pains in the limbs, sometimes with vomiting, occasionally with a chill. Pain on mastication, or sore throat and difficulty in swallowing, are present in some cases. Very soon the gums become spongy and begin to bleed, and with the progress of the disease a general hemorrhagic tendency develops, manifesting itself under the clinical picture of a hemorrhagic purpura, with petechiae or ecchymoses in the skin, and hemorrhages from the mucous membranes.

Though the cervical lymphnodes may be slightly enlarged, there is usually no general lymphnode enlargement in the early stages of the disease. The tonsils, however, are often enlarged and reddened, and may be covered with exudate. As the disease progresses, there is often a gradual moderate enlargement of all the peripheral lymphnodes, but this may not become notable before death occurs. The spleen may not be palpable in the beginning, but later becomes enlarged to a variable extent. The urine usually shows the characteristics of a tubular nephritis.

FIG. 242



Acute lymphatic leukemia in a girl of 3½ years, showing enlargement of cervical and axillary lymphnodes, and spleen, and slight enlargement of the liver

THE BLOOD.—The chief abnormality involves the white corpuscles. At the beginning the white count may be normal, the so-called aleukemic stage. Within a few days, the white count runs up to from 50,000 to 200,000, and may even reach 500,000. With the increase there is a remarkable transformation in the differential count, nearly all the corpuscles being mononuclear basophiles. The lymphocytes usually reach 90 to 98 per cent, and the large lymphocytes are numerous, and usually are the predominant form. The remaining 2 to 10 per cent are mainly polymorphonuclear neutrophiles, with a few eosinophiles. Myelocytes are usually absent.

The red corpuscles are normal in number and appearance in the early stage. With the progress of the disease a severe anemia develops, the hemoglobin falling to 20 to 40 per cent, and the red count to 1,000,000 to 3,000,000. The color index may be normal or slightly reduced. Nucleated red cells may appear in the blood when the anemia becomes severe, but they are not numerous.

DIAGNOSIS.—The diagnosis depends on the examination of the blood. The disease cannot be recognized in the initial aleukemic stage, unless a notable relative lymphocytosis precedes the increase in the white count. The changes in the blood should be pronounced by the end of the first week at the latest.

Whenever the physician is confronted with a disease of acute febrile onset resembling that of an acute infection, especially if associated with stomatitis, tonsillitis, and the rapid development of a tendency toward hemorrhages, he should remember the possibility of acute lymphatic leukemia. If an examination of the blood shows a marked increase in the white count, with a marked relative increase in the lymphocytes, or if even without a marked increase in the white count, the differential count shows a lymphocytosis of 90 per cent or over, a diagnosis of acute leukemia can be made.

The diseases especially to be differentiated by means of the blood examination from acute leukemia are the following: (1) Acute infections in general; (2) stomatitis and tonsillitis in particular; (3) the hemorrhagic diseases, including scurvy and purpura.

A differential diagnosis between acute lymphadenoid leukemia, and acute myeloid leukemia cannot always be made. In the latter form the predominant cell is often not the myelocyte, but a cell which, although of myeloid origin, resembles the ordinary large lymphocyte in appearance under ordinary staining methods. (Naegeli's myeloblasts.) The distinction between these myeloblasts and lymphoblasts can only be made by an expert hematologist with special staining methods. For this reason many cases have been reported as acute lymphatic leukemia which were really cases of acute myeloid leukemia. The distinction is not necessary for purposes of prognosis and treatment, and in children it is best to class all cases showing the clinical picture described above as acute lymphadenoid leukemia, recognizing that the autopsy may occasionally disclose the lesions of the myeloid form.

PROGNOSIS.—The prognosis of acute lymphadenoid leukemia is bad, the disease apparently being always fatal. Death occurs from exhaustion following the hemorrhages and progressive cachexia. The duration of the acute form varies from a few weeks to two months.

Cases in which the disease runs a course longer than two months should be considered as chronic rather than acute.

TREATMENT.—The treatment of all forms of leukemia will be considered together.

CHRONIC LYMPHADENOID LEUKEMIA

(Chronic Lymphatic Leukemia; Chronic Leukemic Lymphadenosis)

This form is very uncommon in early life.

SYMPTOMS.—The onset is insidious. The first thing noted in most cases is that the patient is growing pale, becomes disinclined to exertion, and complains of feeling weak. He may complain of some dyspnea and palpitation. The parents may also note enlargement of the cervical lymphnodes.

On physical examination one finds a general lymphnode enlargement, involving the cervical, axillary and inguinal regions. The enlargement is usually not great, the nodes varying from the size of beans to that of hickory nuts. They are fairly firm, a little sensitive to pressure, and are never adherent to the skin. The spleen is palpable and sometimes is fairly large, but never reaches anything like the size met with in the chronic myeloid form of the disease. It feels firm and smooth, and the notch often cannot be made out. The liver shows only a slight enlargement. Some cases are seen in which the enlargement of the peripheral lymphnodes and spleen are not notable, the hyperplasia affecting chiefly the internal nodes. The temperature may be normal, or there may be slight irregular fever.

THE BLOOD.—The white count is high, averaging between 100,000 and 500,000. The differential count shows that the increase is wholly in the lymphocytes, the small mononuclear basophiles dominating the blood picture. The lymphocytes may reach 90 per cent or over, the white corpuscles of myeloid origin showing a proportional relative diminution, while most of the lymphocytes are of the small type. Larger forms sometimes appear, but the blood picture in chronic lymphadenoid leukemia is more uniform than in any other form of the disease. The hemoglobin and red corpuscles show evidences of a varying degree of anemia with the same characteristics as are seen in the acute form of the disease. Normoblasts are far less common than in chronic myeloid leukemia.

DIAGNOSIS.—The diagnosis depends entirely upon the results of the blood examination. This excludes all other conditions characterized by progressive anemia and cachexia, and by chronic enlargement of the lymphnodes.

PROGNOSIS.—In adults the disease may run a long course marked by exacerbations and remissions. The few cases reported in children suggest that the disease runs a more rapid course in early life, the patients usually dying within a few months. The cause of death is the exhaustion following the progressive cachexia or the development of hemorrhages.

ACUTE MYELOID LEUKEMIA

(Acute Leukemic Myelosis)

This is a very rare form and is almost unknown in early life. A certain number of cases, however, which were diagnosed during life as acute lymphatic leukemia, have proved at autopsy to be of the myeloid type.

SYMPTOMS.—The general symptoms are indistinguishable from those of acute lymphadenoid leukemia (q.v.).

THE BLOOD.—The same increase in the total number of white corpuscles, as is seen in the acute lymphatic form, is seen in most cases. The differential count with ordinary staining methods is indistinguishable from that seen in the acute lymphatic type. Finer staining methods, however, will show that the white cells consist almost entirely of Naegeli's myeloblasts. In other cases the varying percentage of ordinary myeloblasts gives a clew to the myeloid lesion. The anemia shows the same characteristics as in the acute lymphatic type, except that normoblasts are rather more numerous.

DIAGNOSIS.—Some cases of this type cannot be distinguished clinically from acute lymphatic leukemia except through the finer staining reactions brought out by an expert hematologist.

PROGNOSIS.—The course and termination of the disease is the same as in acute lymphatic leukemia.

CHRONIC MYELOID LEUKEMIA

(Splenomyelogenous Leukemia; Splenic Myelogenous Leukemia; Chronic Leukemic Myelosis)

This is the commonest form of the disease, but is chiefly encountered in adults, being rarer in early life than the acute lymphatic form.

SYMPTOMS.—The onset is insidious with progressively increasing weakness and pallor. Sometimes it is these symptoms and sometimes the enlargement of the spleen which first leads to the calling of a physician. There are occasionally various subjective symptoms, such as pain in the left side of the abdomen, dyspnea, palpitation, and digestive disturbance. Sometimes, especially in children, hemorrhages occur comparatively early in the course of the disease. On physical examination there is usually found pallor, some emaciation, and at times petechiae in the skin, or swollen bleeding gums. The most important sign is the marked enlargement of the spleen, which is always felt several fingers' breadth below the costal margin, and in many cases fills up the entire left side of the abdomen, and even may extend beyond the umbilicus. The organ feels hard and smooth, and the notch can usually be made out. It may be somewhat tender if a perisplenitis be present. The liver is usually also notably enlarged and may reach a very large size. There may be some tenderness over the ribs and sternum. There is often a moderate irregular fever, and there may be night sweats.

THE BLOOD.—The white count is high, averaging between 100,000 and 200,000, and sometimes reaching 500,000 or even more. There may be remissions in the disease when the white count is but little increased above the normal. The blood picture seen in stained smears is characteristic, being dominated in the majority of cases by the presence of a large number of neutrophilic myelocytes. There is also an enormous increase in the absolute number of the polymorphonuclear neutrophiles, which is often so great that they maintain their normal percentage in the differential count, averaging 60 to 70 per cent of all the white cells. There is also a great increase in the polymorphonuclear eosinophiles, and in the mast cells or polymorphonuclear basophiles. Eosinophilic myelocytes are also present, and various transitional forms. Lymphocytes are seen in about their normal absolute numbers, but on account of the marked increase in the cells of myeloid origin their relative number is greatly diminished.

The red corpuscles show the characteristics of a developing secondary anemia, their number being normal at the beginning, but later becoming diminished with the presence of imperfectly formed cells and normoblasts.

DIAGNOSIS.—The diagnosis depends entirely upon the results of the blood examination. The great increase in the absolute number of white corpuscles with the presence of large numbers of myelocytes and other cells of myeloblastic origin, is necessary for a diagnosis. Such a finding excludes all other conditions characterized by progressive anemia with marked splenic enlargement.

PROGNOSIS.—The disease is eventually fatal, although long remissions may occur. The average duration in adults is from two to four years. Death occurs from general exhaustion, from terminal infection, or from hemorrhage. Too much hope should not be built upon the remissions, which are often followed by exacerbations of a more acute type.

TREATMENT OF LEUKEMIA

There is at present no evidence that any form of treatment can obtain more than a temporary amelioration of the symptoms. The apparent effects of treatment are more marked in the chronic than in the acute forms.

Three methods of treatment have been widely advocated in leukemia. These are (1) the administration of arsenic; (2) treatment with the Roentgen ray; (3) the administration of benzol. There are reports of temporary improvement following the use of all three methods of treatment. In considering the value of such reports it must not be forgotten that leukemia, especially in its chronic forms, often shows spontaneous temporary remissions. The evidence in favor of the value of arsenic and of X-ray treatment is less striking than that in favor of the use of benzol, although many cases of temporary improvement under radiotherapy have been reported. The majority of roentgenologists at the present time do not recommend the use of this treatment as holding out much hope of benefit. There is no reason, however, why radiotherapy should not be tried. The administration of benzol appears to be the method of treatment which gives the best prospects of temporary amelioration of the symptoms. Indeed, a certain number of cases have been reported as cured, but the interval following the apparent cure has been too short to permit the drawing of a final conclusion. There is no doubt, however, that benzol will in the majority of chronic cases, and in some acute cases, cause a marked reduction in the number of white cells in the blood, and this reduction of the white cells is accompanied by all the evidences of temporary improvement in the general condition. Benzol appears to act directly on the hematopoietic tissues, although the physiological action of the drug is not fully understood. There is no doubt that benzol in improper dosage may have a dangerous toxic action. Furthermore, a number of writers have brought out the fact that the reduction in the number of white corpuscles caused by the giving of benzol may go too far and may be accompanied by dangerous symptoms. The reduction does not cease immediately when the benzol is omitted. For this reason benzol should be given very cautiously, the patient being under the closest supervision as to his general condition, and daily examinations of the blood should be made. Many writers believe that the giving of benzol should be omitted when the white count appears to show a progressive fall. At any rate, the administration of benzol should not be continued after the white count has fallen to 25,000.

The dose of benzol which is safe and yet which will produce the desired effect, is still a subject of discussion. The preponderance of evidence seems to show that doses of 3 minims three times daily, are safe to begin with in children, and that if the desired effect is not produced, the dosage may be cautiously increased, with careful watching of the urine and of the general condition of the patient. There is evidence that benzol will produce temporary improvement not only in the blood, but also a diminution in the size of the spleen or lymphnodes, and that cases which have resisted X-ray treatment will sometimes improve under benzol therapy. There is no doubt that benzol may be a somewhat dangerous remedy, and its final value is still under discussion. The remaining treatment of leukemia is wholly symptomatic, and resembles that of anemias of the same severity. Transfusion may be of value when hemorrhages develop.

PROBLEMS AND RESEARCH

The problem of the etiology of leukemia is being extensively studied. Some writers believe that the disease is best classified with the sarcomas. The tendency of the last few years, however, is to regard leukemia as a manifestation of infection. Some investigators believe that it represents an infection *sui generis*, while others believe that the disease is a sequel or accompaniment of established infections. In favor of the first view are some reports of the isolation from the lesions of the disease of a peculiar diphtheroid microörganism resembling that which occurs so constantly in Hodgkin's disease. Simon and Judd have reported such a finding in acute leukemia, and Steele has reported a similar finding. In favor of the second view are reports of the cultivation of various known microörganisms, including staphylococci and streptococci, in cases of leukemia. As, however, the lymphnodes and spleen frequently contain such organisms, these findings are by no means conclusive evidence of an etiological relationship.

One difficulty which has attended the establishment of an infectious origin for leukemia has been this, that inoculation of human material into animals has been followed by negative results. Von Hanseman believes leukemia to represent a manifestation of infection like gumma, which is not inoculable. Successful inoculation experiments have been carried out with the virus of chicken leukemia. Research is also being carried out with studies of the agglutinating power of the blood in leukemia toward various microörganisms. Metabolism studies in leukemia are also being carried on.

The status of the benzol treatment has already been discussed. It has certainly not proved to be all that it promised, and much further knowledge is necessary before we can assign to benzol its final place in the treatment of the disease.

CONGENITAL FAMILIAL HEMOLYTIC ICTERUS

This is a rare disease. It is hereditary and in some instances has been traced through several generations. The few autopsies which have been made show no other notable lesions than a simple hypertrophy of the spleen. The principal symptoms are jaundice and splenic enlargement. The jaundice is a true hemolytic icterus, not merely a pigmentation of the skin. In addition to jaundice there is moderate anemia, urobilinuria, and slight enlargement of the liver. The disease lasts throughout life without much effect upon the general condition. Although rare, it must be considered when an apparent icterus neonatorum exists, and also in the differential diagnosis of diseased conditions with splenic enlargement.

II. DISEASES OF THE SPLEEN

ENLARGEMENT OF THE SPLEEN

(The Splenomegalies)

Microscopic lesions of the spleen both of toxic and of infectious origin are frequently found post-mortem in many recognized diseased conditions. From the clinical point of view the only lesion of the spleen of importance is enlargement. Enlargement of the spleen is particularly common in early life, especially in infancy. Most of the splenomegalies do not represent a primary disease of the spleen, but represent lesions which are secondary to well recognized diseases. Splenomegalies may be acute or chronic. In the acute splenomegalies the enlargement is due partly to hyperemia and partly to hyperplasia of the splenic pulp. The principal diseases in which acute splenomegaly is seen are certain of the acute infections, particularly typhoid fever and malaria. A moderate degree of acute splenic enlargement is also seen at times in general sepsis, ulcerative endocarditis and pneumonia. In all these cases the spleen is rather soft to palpation, and it may show a notable change in size from day to day.

Chronic enlargement of the spleen is also a very common manifestation of infections or intoxications of known etiology. It is also a common manifestation in the diseased conditions primarily affecting the blood, which have just been described. The conditions in which enlargement of the spleen is seen most often in early life are the following: 1. In rickets. 2. In certain of the severer forms of anemia. 3. In leukemia. 4. In Hodgkin's disease (pseudoleukemia). 5. In chronic passive congestion due to diseases of the heart and lungs. 6. In congenital syphilis. 7. The amyloid spleen seen in connection with amyloid disease of the liver and kidneys in chronic suppurative processes. 8. In certain cases in which the spleen is involved in tuberculosis. 9. In congenital and familial hemolytic jaundice.

Rarer causes of splenic enlargement are echinococcus cyst of the spleen, the tropical splenomegaly of kala-azar, and the ague cake, which follows repeated malarial infections. In all these conditions, the splenic enlargement is merely a symptom of the underlying disease, and the recognition of its cause depends upon the diagnosis of the primary affection.

There are two types of chronic splenomegaly which are generally considered to represent primary diseases of the spleen itself. These conditions, though rare, are sometimes seen in children. They are Banti's disease and splenomegaly of the Gaucher type.

BANTI'S DISEASE

(Primary Splenomegaly with Secondary Hepatic Cirrhosis; Primary Splenomegaly with Anemia)

This condition has been confused both with the anemias with splenic enlargement, and with the pseudoleukemias. It is differentiated from the former by the fact that in Banti's disease there is every evidence that the primary lesion is in the spleen rather than in the blood, or in any other part of the blood-making apparatus. The lesion in the spleen does not differ from that seen in the von Jaksch's type of anemia, being a simple hypertrophy. For this reason cases of Banti's disease occurring in infancy have often been confounded with von Jaksch's anemia, although there is every evidence that the etiology is wholly different, and that Banti's disease represents a primary affection of the spleen. Banti's disease is differentiated from the pseudoleukemias in that the lesion of the spleen is a simple hyperplasia and does not show the peculiar microscopic anatomy characteristic of Hodgkin's disease. Although rare, Banti's disease is sometimes seen in early life, and its recognition is peculiarly important for the welfare of the patient, because if a correct diagnosis be made in the early stage of the disease, splenectomy may be performed, resulting in a complete cure.

ETIOLOGY.—The cause of Banti's disease is unknown. It is probable, as stated above, that the condition is primarily a disease of the spleen, leading to an intoxication that causes a secondary cirrhosis of the liver.

PATHOLOGICAL ANATOMY.—In the cases that have come to autopsy, the spleen has shown the ordinary lesion of chronic hyperplasia with fibrous thickening of the splenic pulp and atrophy of the Malpighian bodies. There is also a moderate grade of atrophic cirrhosis of the liver.

SYMPTOMS.—The disease develops gradually and its progress is usually described in three stages. In the first stage there is a gradually increasing splenic enlargement, at first without any other symptoms. Later, the blood shows the characteristics of a secondary anemia, with diminution of the number of red corpuscles, and low color index, and either no marked change in the white corpuscles, or else a leukopenia with a relative lymphocytosis. The liver in this stage may show a slight enlargement, but there is usually no jaundice.

The second stage is characterized by the development of jaundice and gastro-intestinal symptoms. The anemia increases rapidly, the patient loses weight and a progressive cachexia begins.

In the third stage the most prominent symptom is the ascites, caused by the secondary cirrhosis of the liver. There are also apt to be hemorrhages from the esophagus or intestines, and cachexia becomes very marked, the patient eventually dying of exhaustion or from hemorrhage.

DIAGNOSIS.—The recognition of Banti's disease is particularly difficult in infancy and early childhood. In older patients, anemic conditions are not so likely to be characterized by marked splenic enlargement, and consequently even in the early stage of the disease, before the characteristic jaundice and ascites have developed, the combination of anemia with splenic enlargement is very significant. In young children, however, the type of anemia described as anemia infantum pseudoleukemica closely resembles Banti's disease. It is distinguished from Banti's disease in that in the latter the splenic enlargement precedes the development of a severe anemia, and is often marked before the blood shows any very notable changes. Nevertheless, cases of Banti's disease in young children can often not be distinguished from the anemias with splenic enlargement until the later stages of the disease have developed, and in these later stages splenectomy as a means of cure does not present so favorable an outlook. It should be remembered in this connection that anemia with splenic enlargement is a very common syndrome in early life, being seen not only in anemic conditions of unknown cause, but also in rickets and other conditions of known cause associated with anemia. Banti's disease, on the other hand, is very rare at this age. It is best therefore to consider cases of anemia with splenic enlargement occurring in the first three or four years of life to be due to causes affecting the blood rather than to causes affecting the spleen, unless the clinical picture of Banti's disease with marked splenic enlargement preceding the anemia is very pronounced.

It is sometimes difficult to distinguish Banti's disease from primary cirrhosis of the liver with secondary stasis of the spleen. Both conditions are rare in early life. In primary hepatic cirrhosis ascites is an earlier symptom, and anemia and cachexia are not so pronounced. Myeloid leukemia is easily ruled out by examination of the blood. Other diseased conditions to be excluded in making a diagnosis of Banti's disease are the various secondary splenomegalies enumerated above, particularly the splenic enlargement of congenital syphilis, that of tuberculosis, and that of Hodgkin's disease.

PROGNOSIS.—The outlook in Banti's disease is unfavorable without proper treatment.

TREATMENT.—In Banti's disease splenectomy should be performed.

On account of the comparative seriousness of the operation, great care should be used in making the diagnosis. All other possible causes of splenomegaly should be carefully excluded. There is, of course, a certain amount of immediate operative risk attending splenectomy. If, however, the patient survives the operation, the outlook for permanent cure is extremely good, particularly if the operation be performed in the earlier stages of the disease.

PROBLEMS AND RESEARCH.—The principal problem connected with Banti's disease is that of etiology. The investigations now being made are similar in character to those which are being carried out with the leukemias and pseudoleukemias.

GAUCHER'S TYPE OF SPLENOMEGALY

This condition has often been described as a variety of Banti's disease, but it is probable that it is wholly different in etiology and pathogenesis.

ETIOLOGY.—The cause of the condition is unknown. It is very rare, and there are some instances pointing to heredity as a factor. There is no connection with tuberculosis or syphilis.

PATHOLOGICAL ANATOMY.—The enlargement of the spleen is due to the accumulation of very large cells resembling endothelial cells. The latest studies suggest that these cells are of connective tissue rather than of endothelial origin. As similar groups of cells are found in the liver, bone-marrow and lymphnodes, it is probable that the disease involves the whole leukopoietic system, as does leukemia.

SYMPTOMS.—The disease is characterized clinically by chronic splenomegaly, enlargement of the liver, moderate anemia, and a peculiar brownish or grayish discoloration of the skin.

DIAGNOSIS.—The condition is distinguished from Banti's disease by its hereditary features and by the pigmentation of the skin. In Addison's disease there is neither splenomegaly nor enlargement of the liver.

PROGNOSIS.—The disease is much less progressive and fatal than is Banti's disease.

TREATMENT.—None is indicated unless definite symptomatic indications arise.

III. DISEASES OF THE LYMPHNODES

In early life as compared with adult life, the lymphoid tissue is relatively well developed. This is particularly the case with the tonsils and adenoids, but is also true of the external and internal lymphnodes and of the lymphoid follicles in the intestine and in other parts of the body. This relative good development of the lymphadenoid tissue is so marked a peculiarity of early life that it becomes difficult at times to draw the line between the normal and the abnormal. Furthermore, the degree of development of the lymphadenoid tissue varies in individual children. Certain children have from birth a development of lymphadenoid tissue, which compared with the average is excessive. This congenital lymphadenoid over-development is a constitutional peculiarity of the individual child, and heredity is a common etiological factor. Frequently the parents of such children showed in early life the same peculiarity, and it is not uncommon to find a relative overdevelopment of the lymphnodes in all the children.

Another peculiarity of the lymphadenoid tissue in early life is a tendency toward hyperplasia from comparatively slight causes. This also is a normal condition peculiar to early life and is most marked in infancy, becoming progressively less as the child grows older. Individual children also show variations in this tendency toward lymphadenoid hyperplasia from slight cause, and these variations also represent constitutional peculiarities in which heredity plays an important part. In infancy and early childhood, the lymphnodes connected with the gastro-intestinal and the bronchial mucous membranes are particularly susceptible to the causes which produce hyperplasia. As the child grows older, retrograde changes occur in the various groups of lymphnodes, causing a diminution in the size of the nodes, and with it a diminution in the tendency toward enlargement from comparatively slight causes. The lymphnodes connected with the digestive tract, which are situated in the mesentery and in the retroperitoneal tissues, are the first to diminish, the retrograde changes usually beginning after the second year, the relative enlargement disappearing by the sixth year. The bronchial group of lymphnodes are next in order in undergoing retrograde change. Consequently in later childhood it is the tonsils and other lymphnoid tissue of the pharynx and the cervical lymphnodes which show the greatest relative enlargement and which are most susceptible to further enlargement from various causes. These groups of lymphadenoid tissue do not begin to undergo retrograde changes until after the seventh year, the adult condition not being reached until about the age of puberty.

ACUTE LYMPHADENITIS

This is a condition in which an acute inflammatory reaction takes place in the lymphadenoid tissue.

ETIOLOGY.—The cause of acute lymphadenitis is *infection.* The inflammatory changes in the lymphadenoid tissue may be caused by the presence in the tissue of the infecting microörganisms, or by the bacterial toxins brought to the lymphadenoid tissue by the lymphatics which drain the infected area. When the process is caused by the actual presence of bacteria, the microörganisms enter through the mucous membrane or skin and are brought to the lymphnodes by the lymphatics. In the majority of cases the infection of the lymphnodes is secondary, the primary site of the infection being the mucous membrane or the skin. In some cases, however, the infection of the lymphnodes is primary, the microörganisms reaching the lymphadenoid tissue without causing any lesions of the mucous membrane or skin at the point of entrance. When the acute inflammation of the lymphadenoid tissue is due to toxins without the actual presence of bacteria, the process is always secondary to the infectious lesions in the region drained by the lymphatics leading to the lymphnodes involved.

Acute lymphadenitis occurs most often in connection with infections of the mucous membrane, particularly that of the pharynx. It is therefore the cervical lymphnodes which are most often involved. Acute cervical lymphadenitis is a common complication of such infections as diphtheria, scarlet fever, measles and influenza, in all of which diseases the pharynx is the seat of inflammation. It is also seen accompanying the acute catarrhal infections of the tonsils and pharynx. It is also seen in connection with lesions of the mouth, such as ulcerative stomatitis or carious teeth, and in connection with infections of the ears, nose and eyes. It is sometimes seen in connection with eczema and with infectious lesions of the scalp. Acute axillary lymphadenitis may occur with any infectious lesion of the upper extremities, such as vaccination, or septic infection of the hand. Acute inguinal lymphadenitis is most often due to an infection of the external genitals, particularly vulvovaginitis and balanitis. It is also seen in localized septic infections of the skin of the lower extremities.

Acute lymphadenitis of the internal lymphnodes, notably the bronchial and mesenteric groups, is also not uncommon in connection with infections of the mucous membrane of the lungs or intestine. Acute inflammation of these internal lymphnodes, however, is not often to be recognized clinically, as it presents no symptoms which can be distinguished from those of the primary disease.

The microörganisms most commonly associated with acute lymphadenitis are the streptococcus, staphylococcus, pneumococcus, and the bacillus of influenza.

PATHOLOGICAL ANATOMY.—The inflammatory process in the lymphnodes may be non-suppurative or suppurative, according to the character and virulence of the infection and the susceptibility of the lymphadenoid tissue. Suppuration can only occur when the bacteria are actually present in the lymphadenoid tissue. In the non-suppurative cases, which can occur either from the presence of bacteria or from the absorption of toxins, the changes are confined to acute congestion, serous exudation, and active hyperplasia of the lymphadenoid elements, with some exudation of polymorphonuclear or of endothelial leukocytes. The process terminates by resolution. In suppurative lymphadenitis, the lymphnode becomes the seat of an abscess, which eventually tends to open externally. There is usually also considerable inflammation in the surrounding cellular tissue.

Suppurative inflammation is more common in infancy than in later childhood. In older children, suppurative lymphadenitis occurs most frequently in connection with scarlet fever, is sometimes seen with diphtheria, but rarely occurs in connection with the ordinary catarrhal inflammations of the pharynx and tonsils. Suppurative lymphadenitis is usually unilateral. Non-suppurative lymphadenitis may be either unilateral or bilateral.

SYMPTOMS.—In non-suppurative lymphadenitis there is very little or no constitutional disturbance. The nodes become acutely enlarged, and are hard and tender. There is no reddening of the skin, and fluctuation is not present. There may be some fever. The swelling usually subsides slowly in a few weeks. In suppurative lymphadenitis, the most typical cases of which occur in infancy, there are present at first the symptoms of the original disease. Acute lymphadenitis manifests itself first by the appearance of a tumor. The nodes most frequently affected are those of the deep cervical group, and the tumor appears most often just below the angle of the jaw at the anterior border of the sternocleidomastoid muscle. In the acute specific infections the tumor appears during the height of the disease, increases rapidly in size, and quickly shows signs of softening. When acute suppurative lymphadenitis accompanies the catarrhal infections of the throat in infancy, the swelling is not so rapid, but continues after the catarrhal process in the throat has begun to subside. The tumor is at first hard and tender, and if there is marked inflammation of the surrounding tissue, there may be pain and heat. The tumor increases until it reaches the size of a walnut or of a hen's egg. Fluctuation usually appears by the end of the second week, but sometimes is not seen until the third or fourth week. There may be reddening of the skin over the swelling.

In the majority of cases there is only moderate fever and very little constitutional disturbance. In some cases, however, particularly in young infants, there may be high fever ranging from 102° F. to 104° F., with marked constitutional symptoms, and sometimes with severe prostration. The symptoms of axillary and of inguinal lymphadenitis are similar to those of the more common cervical form.

DIAGNOSIS.—The recognition of acute lymphadenitis is very easy. The acuteness of the process with the tenderness and other signs of an inflammatory reaction differentiate the condition from most other enlargements of the cervical lymphnodes. The disease is most frequently mistaken for mumps, in which also an acute tender swelling in the cervical region occurs. With careful examination, however, the differentiation from mumps is not difficult. The swelling in mumps extends up in front of the lobe of the ear, a condition which is never seen in lymphadenitis.

It is necessary not only to recognize the disease as an acute inflammation of the cervical lymphnodes, but also to distinguish the suppurative from the non-suppurative form. When fluctuation on palpation of the tumor can be detected, there can be no doubt as to the diagnosis, as no other condition produces so acute an inflammatory reaction with softening. In the majority of cases, however, even when suppuration has occurred, it is deep in the tissues, and the surrounding inflammatory induration is so great that fluctuation cannot be detected. It is important to remember in this connection that apart from the specific infections, acute suppurative cervical lymphadenitis is essentially a disease of infancy. Evidences of involvement of the surrounding cellular tissue point toward suppuration. The presence of marked fever or of constitutional disturbance is also significant.

PROGNOSIS.—The prognosis of acute lymphadenitis, whether suppurative or non-suppurative, is good. There is little or no tendency for the infection to extend into other tissues, or to cause a general sepsis. In the non-suppurative cases the swelling subsides in from four to six weeks, but a small tumor may persist for several months. In suppurative cases, the abscess, after being opened, heals rapidly, a permanent sinus being rare, and complete recovery occurs in about the same time as in the cases terminating by resolution.

TREATMENT.—All acute catarrhal infections of the mucous membranes should receive proper treatment as a prophylactic measure against acute lymphadenitis. The measures used are careful syringing, and pharyngeal or nasal spraying.

In the stage of acute inflammation, before it has become evident whether or not suppuration has occurred, the ice bag should be kept continuously applied to the swelling. It is doubtful whether this measure has much influence in preventing suppuration, but it is useful for the relief of pain. In some cases young children dislike and resist the ice bag, and under such circumstances it is not worth while to insist on its use. The use of "antiphlogistine" makes a great impression of benefit on the child's parents, but does no real good. The same is true of painting with iodin, or of inunctions with the oleate of mercury or any other ointment.

When the progress of the case suggests that suppuration has begun, hot applications should be used instead of the ice bag for the relief of pain. It is probable that this procedure hastens the pointing of the abscess.

Except in certain exceptional cases with constitutional disturbance severe enough to be threatening, incision should be deferred until the abscess points. If this is done, refilling is rare, and the abscess heals promptly under proper aseptic treatment. Curetting may be necessary in rare cases with much broken down tissue.

In non-suppurating cases due to carious teeth, eczema, ulcerative stomatitis, or balanitis, treatment of the primary causes produces a rapid subsiding of the swelling.

CHRONIC LYMPHADENITIS

A generalized enlargement of the lymphnodes is very common both in infancy and childhood. It represents a very typical reaction to disturbance of the general nutrition. It is so common as to be practically universal in children reared under faulty hygienic surroundings, whether in institutions or in crowded tenements. It may be caused by anything which produces malnutrition, or lowers the vitality of the tissues. It is seen in chronic gastro-intestinal disease, in anemia, and in rickets.

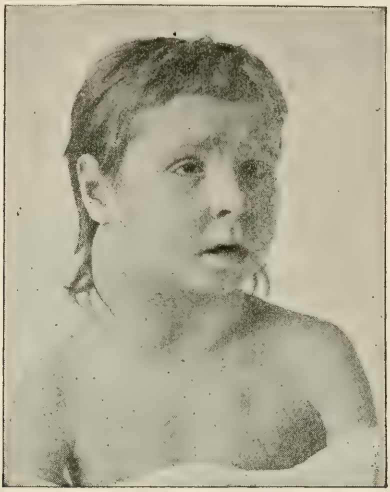
It is not to this generalized hyperplasia of the lymphadenoid tissue that the term chronic lymphadenitis is here applied, but to more localized lymphnode enlargement involving particular groups of nodes. Both the external and the internal lymphnodes may be involved in this process, but chronic lymphadenitis of the internal lymphnodes, unless tuberculous, cannot often be recognized clinically, and only the external groups will be considered.

The condition is less common than acute lymphadenitis, and is seen mainly in infants and young children. The process is a simple hyperplasia, which is neither syphilitic nor tuberculous. It is usually the cervical lymphnodes which are involved.

ETIOLOGY.—The enlargement is due to the absorption of irritating products from some area of inflammation drained by the lymphatics involved. It may follow one or several attacks of acute lymphadenitis. It is often due to chronic inflammation of the mucous membrane or skin. It is seen with chronic pharyngitis, diseased adenoids or tonsils, eczema, and pediculosis of the scalp.

SYMPTOMS.—The nodes on both sides of the neck are usually involved, usually a group rather than a single node. The swelling is less than in acute lymphadenitis, and less than in most cases of tuberculous lymphadenitis. The swelling increases slowly for a few months, remains stationary for a few months more, then very slowly subsides. There is no tendency to suppuration or caseation, no tenderness, and no constitutional disturbance.

FIG. 243



Chronic cervical adenitis

DIAGNOSIS. *Tuberculous Lymphadenitis.*—Simple chronic lymphadenitis is most frequently confounded with tuberculosis. The chief points in the differential diagnosis are found in its usual occurrence in very young children at an age when tuberculosis of the cervical lymphnodes is uncommon, in the evidence of a primary cause to which the chronic lymphadenitis is secondary, in the absence of caseation, suppuration and periadenitis, and finally, in the greater influence of general tonic treatment. The nodes in simple lymphadenitis are movable, and do not become adherent either to the skin or to the deeper tissues. A positive tuberculin reaction can usually be obtained in tuberculous lymphadenitis.

Hodgkin's Disease.—Simple chronic lymphadenitis is distinguished less easily from Hodgkin's disease, but in the latter the enlargement of the nodes is greater and more generally distributed, the secondary anemia is more pronounced, and the disease is of rare occurrence as compared with chronic lymphadenitis. In doubtful cases, excision and examination of a portion of the enlarged node will settle the question.

Syphilitic Lymphadenitis.—The absence of other signs of late syphilis, and the negative Wassermann reaction, make the exclusion of syphilitic lymphadenitis comparatively easy.

New Growths.—New growths are usually to be distinguished by their more rapid growth and greater size and by the absence of an exciting cause, more positively by the microscopic examination of a portion of the node excised for the purpose of diagnosis.

TREATMENT.—In the treatment of simple chronic lymphadenitis, the primary exciting cause should be removed if possible. Local applications should be avoided, and general tonic treatment instituted by means of cod-liver oil, Fowler's solution, the iodid of iron, good food, and out-of-door life.

SYPHILITIC LYMPHADENITIS

The enlargement of the lymphnodes usually seen in congenital syphilis is slight in degree, and generalized in distribution. It differs in no way from the generalized lymphnode hyperplasia which is so common as a result of malnutrition. Occasionally, however, there is a more marked enlargement of the cervical lymphnodes. This is only seen in the late stage of childhood. The recognition of the cause of the lymphnode enlargement depends upon the finding of other signs of late congenital syphilis, such as keratitis, periostitis, and Hutchinson's teeth. The diagnosis is confirmed by the presence of the Wassermann reaction and the rapid subsiding of swelling which takes place under antisyphilitic treatment.

TUBERCULOUS LYMPHADENITIS

Tuberculosis of the internal lymphnodes, bronchial or mesenteric, has already been considered in the general description of tuberculosis in Volume I. It is these forms of lymphnode tuberculosis which are particularly characteristic of the disease in infancy and early childhood.

Tuberculosis of the external lymphnodes is rare in infancy, but is an extremely common manifestation of the disease in later childhood. It is seen most often between the ages of three and twelve years, and in the great majority of cases it is the cervical lymphnodes which are involved.

ETIOLOGY.—Tuberculosis of the cervical lymphnodes may be primary, that is, it may represent the only tuberculous lesion of the body, or it may be secondary to a tuberculosis acquired in infancy, in which the bronchial lymphnodes were the ones first involved. The relation of tuberculous cervical lymphadenitis to chronic tuberculosis acquired in infancy is shown in the diagram on page 704 of Volume I. In such cases, as will appear from the diagram, the manner by which the infection extends from the lungs to the cervical lymphnodes, is through the coughing up of tuberculous material which infects the tonsil or pharynx, and from there is conveyed by the lymphatics to the cervical lymphnodes. It is probable, however, that the majority of cases of tuberculous cervical lymphnodes occurring in later childhood represent the primary stage of the disease. As in all forms of tuberculosis, there is a primary lesion at the point of entrance, usually in the tonsil, but the lesion cannot often be detected either antemortem or post-mortem by macroscopic examination. One evidence that tuberculous cervical lymphadenitis with a microscopic lesion at the point of entrance represents the primary lesion of the disease, is the fact that bovine tubercle bacilli have been found associated more frequently with this form of tuberculosis than with any other. This fact points toward infected milk as being a common cause of tuberculous lymphadenitis. Nevertheless, the human strain of tubercle bacillus is often found, and in these cases the source of the infection is probably contact with tuberculous material coming from an adult consumptive, which is conveyed to the throat of the child.

PATHOLOGICAL ANATOMY.—The lesions found in the cervical lymphnodes are similar to the lesions of tuberculosis in general, and do not require detailed description. Sometimes one group, sometimes another, is first involved, but these variations are not clinically important. The most important point in connection with the pathological anatomy is the fact that the tuberculous process shows two rather distinct forms. In one form, the formation and conglomeration of tubercles in the lymphadenoid tissue takes place more rapidly than in the other, tending to early caseation and softening. There is also considerable inflammatory reaction both in and around the involved nodes, the products of the inflammation being mainly cellular with much less new formation of fibrous tissue. This surrounding inflammation causes the nodes to become adherent to the skin or to other surrounding tissues, and there may even be a perinodular tuberculous abscess.

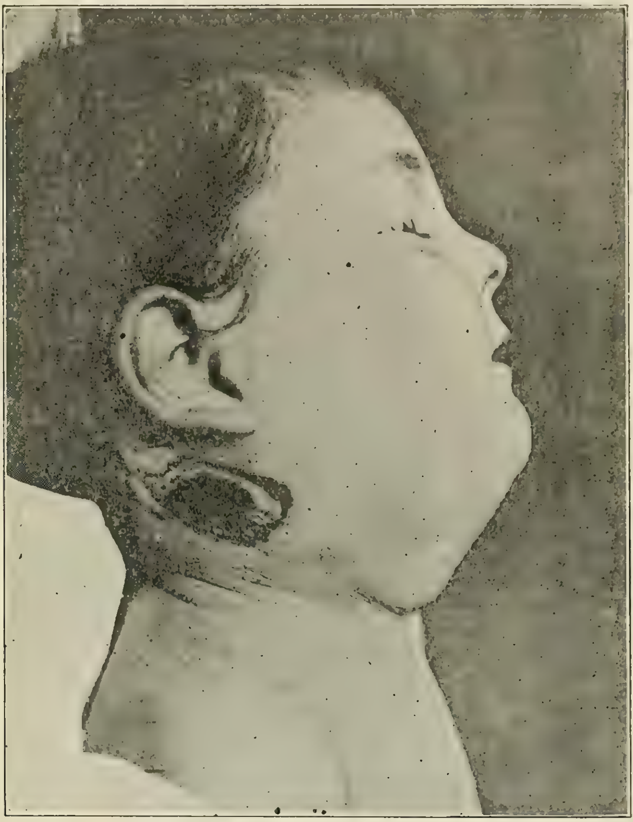
Whether or not the tuberculous process in the lymphnodes takes one of these forms or the other probably depends on the particular resistance developed in the tissues of the individual child. The more chronic, fibrous form represents a higher degree of resistance than does the more rapid suppurating form. In the rapid form, the termination is by extensive breaking down, softening, and eventual external discharge. In the more chronic form, the process may terminate by resolution, and the tuberculous exudate may be absorbed, leaving behind a fibrous cicatrix, or, it may terminate by encapsulation or calcification. At the age when this form of tuberculosis occurs, the general resistance of the body against the spread of the disease is high, and consequently widespread secondary lesions, such as tuberculous meningitis, general miliary tuberculosis, or tuberculous bronchopneumonia, are rare. There is also an increased tendency toward recovery in the infected glands at the age of puberty.

SYMPTOMS.—In the majority of cases the disease begins with a very gradual enlargement of the cervical lymphnodes. In some cases the onset resembles that of acute non-suppurative lymphadenitis, but the swelling, instead of subsiding, persists, and may gradually grow larger. The lymphnode involvement may be unilateral or bilateral. In many cases the process appears to involve both sides at the start, but with the progress of the disease one side outstrips the other, and the advanced changes are usually confined to one side. The gradual enlargement of the nodes is not continuously progressive, but there are remissions and exacerbations. During the exacerbations the nodes increase more rapidly in size, and may be tender and show signs of local inflammation. During the remissions the size of the nodes remains stationary, or may even diminish. In the majority of cases there is no fever, and no general constitutional disturbances. In some cases, however, there is at times, particularly during exacerbations, a moderate febrile reaction with a tendency toward a regular rise of temperature each evening or morning.

The course of the disease varies greatly in different children, being in general more rapid the younger the patient. The duration of the disease between the time of the first appearance of the swelling and the occurrence of suppuration or of retrograde changes, varies from a few months to several years. The tumors attain a variable size, the commonest size being about that of an English walnut, but they may be smaller or much larger. In cases of the fibroid type, the tumors usually continue to be movable and preserve their distinct outline, but the nodes become adherent to one another. In cases which eventually terminate in suppuration, the tumors are at first freely movable with distinct outlines, but later have become adherent, first to each other and to the deeper tissues, and later to the skin. The result is an immovable, irregular, nodular mass, in which it is often very difficult to make out the individual nodes. Even in these cases, softening often cannot be detected for a considerable time, sometimes for months or years. Eventually small spots of softening are found, and later there is distinct fluctuation. Finally the skin over the tumor becomes reddened, and later gives way with a discharge of thick pus. The discharge continues for an indefinite time, until the whole of the tissue involved in the tuberculous process has softened and been thrown off. This same course is repeated with each successive gland that undergoes softening.

The abscess may be confined to the tissue of the node itself, or it may be formed in the tissues immediately surrounding the node. In the former variety the involved node is superficial, has a distinct outline, and is surrounded by little if any evidence of an inflammatory reaction, the area of softening and fluctuation being small, as is the opening when it occurs. In the latter variety there is a more diffuse swelling. The outline of the node may not be made out. The area of softening and fluctuation is larger, and the pus instead of being curdy, is more like that of an ordinary abscess. In still other cases, a deeply situated node breaks down and communicates by a small opening with a more superficial abscess in the cellular tissue. In this last type, the sinus continues to discharge for a long time, until the whole of the deep-lying node has been thrown off, and if healing of the more superficial cavity occurs prematurely, the cicatrix soon breaks down.

FIG. 244



Broken down lymphnode of the neck

When these tuberculous abscesses are allowed to open spontaneously, they form large, irregular ulcers surrounded by undermined and unhealthy looking skin. These ulcers are very intractable and may persist for months in spite of treatment. The scars which they leave behind are very unsightly, with prominent ridges and attached tabs of skin. The areas are of a purplish-red color, are adherent to the deeper tissues, and often undergo cicatricial contractions like the scars formed by burns. They are often also sensitive and painful. They persist throughout life, although they tend to become less noticeable with the lapse of time.

It is very difficult to say what proportion of cases go on to breaking down and softening. Formerly it was supposed that chronic enlargement of the lymphnodes which did not progress to softening was not tuberculous, but we now know that in many cases healing by cicatrization, encapsulation, or calcification, occurs without softening. Other cases may go on for years, and finally breaking down may occur.

Tuberculosis of the cervical lymphnodes has less deleterious effect upon the general health than almost any other form of tuberculosis. The process, though often extensive, is a very local one. Even malnutrition and anemia are not very common accompaniments of this form of tuberculosis. These peculiarities are partly due to the frequency of bovine tuberculosis as a cause of this form of the disease, but are probably mainly due to the comparatively high general resistance which has been attained by children at this age.

DIAGNOSIS.—The cases which present themselves to the physician in a stage when fluctuation or palpable areas of softening are present, are very easily recognized as tuberculous lymphadenitis. Sometimes cases are seen with an unusually rapid course and an early development of suppuration, in which the physician may be in doubt whether he has to do with simple acute lymphadenitis or tuberculous lymphadenitis. Even in the most rapid cases of tuberculous lymphadenitis the process is usually slower than in simple acute lymphadenitis. Furthermore, there is less tenderness, less induration of the surrounding tissues, and less constitutional disturbance. Also, it should be remembered that tuberculous lymphadenitis is rare in infancy, the age at which simple acute lymphadenitis is most common. In the majority of cases, the chronic course with the gradual development of suppuration forms a very characteristic picture.

The cases which present themselves to the physician in the stage before suppuration has occurred, or in which suppuration never occurs, are somewhat more difficult of diagnosis. It is necessary to eliminate other causes of chronic enlargement of the cervical lymphnodes. In simple chronic lymphadenitis the patient is usually younger, and there is usually evidence of a primary cause in the adjacent mucous membrane or skin. The individual nodes are more easily detected, and there is a rapid response to removal of the cause and to general tonic treatment. Evidences of tuberculosis in other parts of the body may be an aid in differential diagnosis. The tuberculin reaction is usually positive in tuberculous lymphadenitis, negative in simple lymphadenitis.

Hodgkin's disease may be difficult to distinguish from non-suppurating tuberculous lymphadenitis. It is very much rarer in children. The enlargement of the lymphnodes is usually not unilateral nor confined to the cervical group, but is apt to be more generally distributed. The nodes in Hodgkin's disease are apt to be somewhat softer and are not matted together as in tuberculosis. Secondary anemia is usually more pronounced. In doubtful cases, Hodgkin's disease is recognized by the examination of a small excised portion of the gland.

PROGNOSIS.—The prognosis of tuberculous cervical lymphadenitis is good. This form of the disease rarely leads to one of the fatal generalized forms of tuberculosis. Furthermore, the tendency is toward eventual complete recovery, and as the age of puberty is approached, the process of recovery is apt to be hastened. On the other hand, the course of the disease is often extremely protracted, one lymphnode after another breaking down in succession for a period of several years. Of course, if there are tuberculous lesions in other parts of the body, the prognosis becomes that of the other forms of the disease.

TREATMENT.—I believe that *tonsillectomy* should be performed in every case of tuberculous cervical lymphadenitis. The tonsils are so frequently the seat of a primary lesion which cannot be detected on ordinary examination, that their removal is always indicated. If they are carefully examined after excision, the small primary lesion can usually be found. In some cases, removal of tuberculous tonsils is rapidly followed by recovery from the process in the lymphnodes. If the nature of the disease is recognized and an early tonsillectomy promptly performed, there is much less tendency toward suppuration. In addition to tonsillectomy, adenoids, carious teeth, and other abnormal conditions in the mouth or pharynx should receive attention.

The *general treatment* of tuberculosis described in Volume I, pages 732 to 735, should be scrupulously carried out. There is some evidence that the *tuberculin treatment* described on page 735, is particularly useful in tuberculous cervical lymphadenitis. Furthermore, some authorities, particularly in Europe, have obtained the most brilliant results by treatment with *heliotherapy,* or the exposure of the diseased parts directly to the rays of the sun.

The particular treatment of the lesions in the cervical lymphnodes is mainly surgical. If after tonsillectomy, and a few months' trial of the general treatment for chronic tuberculosis, the diseased lymphnodes are found to be increasing rather than diminishing in size and number, operation is indicated. Also, when signs of softening have appeared, it is better to operate. The best results are obtained when the operation is done before softening has appeared, and before there is involvement of the skin, or extensive adhesion to adjacent structures. In such cases a thorough operation may be performed, with the removal of the entire chain of lymphnodes, the deep as well as the superficial, and in many cases this procedure will be followed by a permanent cure. In cases which have gone on to the stage of softening, the results are not quite so satisfactory, but nevertheless operation is advantageous in that it shortens the course of the disease, and leaves a small and clean instead of a large and unsightly scar.

If for any reason a radical operation with removal of as much as possible of the diseased tissue is impossible, nevertheless the abscess should be opened as soon as the pus forms in order to prevent the extensive undermining of the skin which otherwise occurs. The opening in such cases should be small, and squeezing of the tissues should be avoided.

Roentgen-ray treatment has been recommended in tuberculous cervical lymphadenitis, and there is evidence that it is of benefit in a certain proportion of the cases. It is most useful in cases in which operation is refused or is contraindicated. The exposure should be short at first, and should not be given oftener than once a week.

HODGKIN'S DISEASE

(Pseudoleukemia; Infectious Granuloma; Malignant Granuloma;
Generalized Lymphadenoma; Malignant Lymphoma)

This is a disease characterized by an infectious granulomatous process in the lymphadenoid tissue with characteristic histological lesions. The disease was formerly considered to be closely connected with leukemia, and there was also considerable doubt whether it represented a distinct pathological entity. At the present time the tendency is to regard the disease as a distinct one with characteristic lesions, and very possibly with a specific cause.

The condition is uncommon in early life. It is almost unknown in infancy, but is found with increasing frequency throughout childhood. It is much more common in males than in females.

ETIOLOGY.—There is considerable evidence that the disease is caused by a specific microörganism which has been named the *corynebacterium granulomatis maligni.* This is a Gram positive non-acid-fast cultivable bacillus. The evidence which associates this bacillus with Hodgkin's disease is the fact that it has been repeatedly found by many observers in the lesions of the disease. Nevertheless, we must admit that the evidence of a causal relationship, though strong, is not yet wholly convincing. Bacteria of various kinds are often found in the lymphnodes as accidental inhabitants, and it is quite possible that this diphtheroid bacillus may be such an accidental inhabitant. On the other hand, it has been found with much greater frequency than has any other microörganism. Furthermore, the lesions of the disease are of such a character as to suggest an infectious process of a character resembling tuberculosis.

PATHOLOGICAL ANATOMY.—The principal lesion is in the lymphnodes, which become greatly enlarged with progressive involvment of fresh nodes. The histology of the lesions is very characteristic. There is proliferation of the endothelial cells, and swollen endothelial cells are found lying in the fibrils of the reticulum. In the interstices of the stroma are found lymphocytes, numerous eosinophiles and large epithelioid giant cells. Similar lymphomatous masses are usually found in the spleen, and may be found in the liver and other organs.

SYMPTOMS.—The principal symptoms are enlargement of the lymphnodes, irregular fever, and secondary anemia.

The onset is usually insidious and the patient is first brought to the physician on account of enlargement of one side of the neck. Examination shows the enlargement to be due to swollen lymphnodes. Later, the nodes on the other side of the neck begin to show enlargement, and thereafter there is progressive enlargement of the nodes in other parts of the body. While it is the cervical lymphnodes which are most often the first to enlarge, the swelling may in some cases begin in other places, such as the axilla, groin, spleen, or mediastinum. The rapidity with which the nodes enlarge as each successive group of lymphnodes is involved is very variable. In certain rare cases it is only the internal lymphadenoid tissue which is involved, and such cases are extremely difficult of diagnosis.

Palpation of the masses of enlarged lymphnodes shows them to be made up of discrete nodules. They may be soft or firm, and usually have an elastic feeling. They are not adherent to one another, to the deeper structures, or to the skin. Softening never occurs. There is a variable degree of enlargement of the spleen.

The irregular fever is quite characteristic. There are periods of very slight elevation of temperature, or in which the temperature may even be normal or subnormal. After such a period there is often a steady rise of temperature lasting from two to four days, when the fever may reach 105° F. The temperature then remains high for several days, and then falls by lysis to the subnormal, when a variable afebrile period occcurs, to be followed by another period of pyrexia.

The blood shows the characteristics commonly associated with a secondary anemia. In the early stages of the disease the number of red corpuscles is unchanged, but later there is a progressive diminution, which continues throughout the disease. The reduction in the hemoglobin corresponds more or less to that of the number of red cells. The white count is unchanged at first, but later there is a moderate increase, the count reaching from 10,000 to 50,000. The differential count shows a normal or relatively increased percentage of polymorphonuclear neutrophiles, a relative and absolute diminution in the lymphocytes, and a relative diminution but absolute increase in the number of eosinophiles. In some cases there is a relative eosinophilia. Other characteristics particularly striking in the early stages of the disease are an increase in the number of large mononuclears, and more especially of transitional forms. A further characteristic is a marked increase in the number of blood platelets.

The course of the disease is shorter in children than in adults, the duration being usually less than three years, and sometimes only a few weeks or months. The patients finally become very anemic and cachectic. Death results from exhaustion or from pressure of enlarged mediastinal lymphnodes upon the respiratory tract. In some cases pressure symptoms from the enlarged internal lymphnodes are prominent. Enlargement of the mediastinal glands may cause cough, dyspnea, cyanosis, dilatation of the superficial veins of the thorax, or dysphagia. Intra-abdominal pressure may cause jaundice, ascites, or edema of the lower extremities. Pressure upon the vessels of the neck or extremities may produce edema.

DIAGNOSIS.—The following are the principal positive points in the diagnosis of Hodgkin's disease. (1) Signs of generalized hyperplasia of the lymphadenoid system; (2) absence of a leukemic blood picture, and the presence of neutrophilic leukocytosis, eosinophilia, and a remarkable increase in the transitional cells; (3) typical histological lesions in a lymphnode excised for diagnosis; (4) the growth of a diphtheroid bacillus in cultures made from an excised node.

In differential diagnosis the conditions to be considered in children are the following: (1) General lymphadenoid hyperplasia; (2) the leukemias; (3) tuberculous lymphadenitis; (4) lymphosarcoma; (5) syphilitic lymphadenitis.

In the general lymphadenitis of nutritional origin so often seen in early life, the enlargement is never so great as to suggest Hodgkins' disease. Furthermore, the characteristic changes in the blood and the characteristic lesion in the excised lymphnode are absent. Leukemia is excluded by the absence of its characteristic blood picture. Tuberculous lymphadenitis is recognized by the tendency of the nodes to coalesce and to break down, the absence of generalized enlargement, the presence of a tuberculin reaction, the absence of Hodgkin's blood picture, and of a characteristic histological picture in an excised node. It must be remembered, however, that some cases of Hodgkin's disease are complicated by tuberculous infection. In lymphosarcoma there is usually a large tumor which invades adjacent structures; both the histological picture and the blood picture are wholly different. Syphilis is rarely to be confused with Hodgkin's disease. It is recognized by the presence of the Wassermann reaction, and of other signs of late congenital syphilis.

PROGNOSIS.—The outlook is bad in early life. I know of no recorded cases of permanent recovery. The disease runs a more rapid course in children, and a fatal ending usually takes place within three years.

TREATMENT.—This is very unsatisfactory, but there is evidence that long periods of remission can sometimes be maintained by some of the measures now in use. The three principal measures of treatment which have given the most evidence of benefit are X-rays, arsenic, and autogenous vaccines. The final value of these methods of treatment has not yet been determined.

PROBLEMS AND RESEARCH.—The chief activity in research on Hodgkin's disease is in the study of the diphtheroid bacillus, which has been isolated from so many cases. Efforts are being made to obtain further proof of an etiological relationship. The value of treatment with autogenous vaccines is being studied. Various immunizing methods are being tested to be used in conjunction with surgical therapy.

IV. DISEASES OF THE DUCTLESS GLANDS

PATHOLOGY OF THE THYROID

The thyroid gland like other tissues is subject to various pathological processes. Among these the ones which have the most clinical interest are those which have to do with the function of the gland. We may have on the one hand an absence or diminution of function leading to the conditions described under such names as cretinism, athyreosis, hypothyroidism, myxedematous idiocy, and infantile myxedema. On the other hand, we may have such conditions of overfunctioning which are described under the names exophthalmic goiter, Grave's disease, and hyperthyroidism.

MYXEDEMA

Myxedema is a constitutional affection, due to lack of thyroid secretion, generally associated with atrophy of the thyroid gland, and characterized clinically by a thickened and dry condition of the skin and subcutaneous tissues, and later by mental failure.

The symptoms are caused by a diminished or total lack of secretion of the thyroid gland due to its atrophic condition. The disease may be *congenital* or *acquired.* Acquired myxedema is seen most commonly in adults, but one form is comparatively common in early life. *Operative myxedema*, or *cachexia strumipriva*, is a condition closely resembling the acquired myxedema of adults, and follows the removal of the thyroid gland by operation. The form of myxedema which is especially related to infants and children is that which is known as *cretinism.*

The term cretinism as generally used applies to cases of myxedema which develop very early in life. Cases of cretinism may upon an etiological basis be divided into two classes. In the first class of cases there is a complete absence of the thyroid gland, the lesion representing a true developmental anomaly occurring during fetal life. It is to this class of cases that the term *sporadic cretinism* is applied. In the second class of cases, the disturbance of the function of the thyroid gland is due to injurious influences acquired in extrauterine life. In certain localities the external injurious influence is evidently something peculiar to a particular geographical distribution, and it is to this group of cases that the term *endemic cretinism* is applied. In other cases, the external injury is of a sporadic nature, and it is to these cases that the terms *hypothyroidism* or *late infantile myxedema* are most properly applied.

It is interesting to note that although sporadic cretinism represents a developmental anomaly which leads to complete absence of the thyroid, yet cretinism is in no way a fetal disease. No sign of the condition is ever noted in the newborn, even when infants are born with a complete absence of thyroid tissue. This is to be explained by the fact that during fetal life the necessary thyroid secretion is supplied to the developing infant through the maternal placenta, and possibly also to a certain extent through the breast during the nursing period.

SPORADIC CRETINISM

(Athyreosis; Congenital Myxedema; Myxedematous Idiocy)

ETIOLOGY AND PATHOLOGICAL ANATOMY.—In this disease the cause, as stated above, is the complete absence of the special tissue of the thyroid gland. The condition should therefore be classified among the malformations, as it represents a true anomaly of fetal development. There is no evidence whatever that the condition is due to injurious influences brought to the fetus from the placental circulation and acting upon a partially developed thyroid. In a certain number of instances, cysts have been found occupying the region of the lateral rudiments of the thyroid, or at the root of the tongue in the region of the isthmus. The arteries which normally supply the gland are also absent, so that the condition appears to be a true primary aplasia. The parathyroids are usually present. There are a certain number of cases in which the lesion represents not a total lack of development of the thyroid, but a hypoplasia of developmental origin. These cases are indistinguishable clinically from the acquired infantile myxedema described below under the term hypothyroidism.

Sporadic cretinism is seen most often in infants of the female sex. There is no evidence of any hereditary factor; usually only one case occurs in a family. There is no evidence as to any causes which might produce this particular developmental anomaly, and its cause must be sought among the general causes which produce such lesions as anencephaly, or the absence of other parenchymatous organs.

SYMPTOMS.—The period after birth at which the symptoms of sporadic cretinism manifest themselves cannot be definitely stated. The period at which the symptoms are first noted varies with the knowledge and acuteness of the observer. When it is the recognition by the parents of something abnormal which causes the child to be brought to the physician, it is usually not until the second half of the first year. The first signs of the disease, however, usually appear much earlier than this, and may often be detected by an acute observer in the early weeks of life in bottle-fed infants, and during the first six months in breast-fed infants. Much depends upon the skill of the observer and upon how carefully the characteristic signs of the disease are sought. The earliest signs are backward growth in length, apathy and failure of mental development, the characteristic facial expression, and the changes in the skin and mucous membranes. The changes in the bone are also early, but at first can only be recognized by means of roentgen ray examination.

THE BONES.—The changes in the bones are probably the most constant and characteristic of all the signs of sporadic cretinism. The principal clinical manifestation of the bony changes is a dwarfing which involves the whole body, but which is first most noticeable in the extremities. All the long bones are shorter and thicker than normal, the extremities appearing stunted and the hands and feet pudgy. Normally at birth the legs are about 43 per cent of the total height, and at four or five years about 50 per cent. In cretins this percentage is lessened to between 35 and 40 per cent of the total length. The base of the nose is broad and flat. The fontanelle remains open often until the eighth or tenth year. Closure of the sutures is often much delayed. Dentition is delayed and irregular, and the second dentition may not begin until adult life. The forehead is often low, and in spite of the open fontanelles the cranial bones may be thickened. The head often appears too large for the body. There is also apt to be spinal curvature, most often a lordosis.

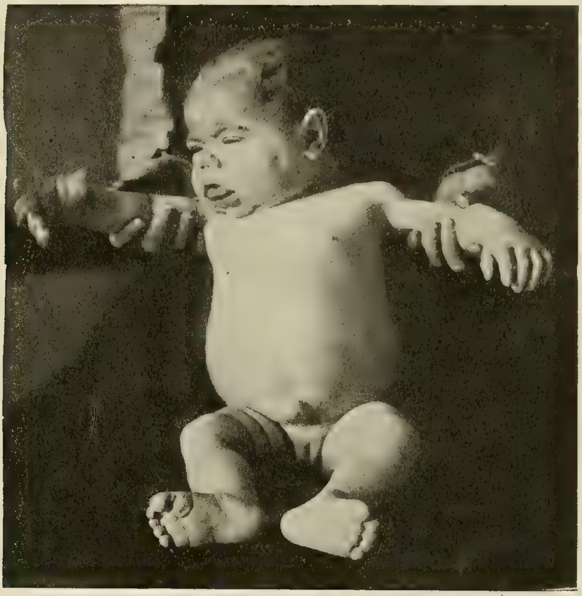
The changes in the bones are due to a disturbance of both the epiphyseal and the periosteal growth of bone. There is a very marked lack of growth of cartilage at the epiphyses, and the normal process by which the capillaries from the bone-marrow extend into the cartilaginous tissue with resorption and the formation of a true bony tissue, is greatly inhibited. The result is the formation of a sclerotic bone, regular in form but reduced in amount. The centers of ossification in the epiphyses appear very late or not at all, and the cartilaginous epiphyseal junction remains for a long time.

Roentgenograms show for the most part the characteristic shortening of the bones, the delayed ossification of the epiphyses, the delayed ossification in the carpal and tarsal bones, and the increased density due to the sclerosis of the shafts of the long bones.

SKIN AND MUCOUS MEMBRANES.—The skin shows a thickening of the subcutaneous tissue, giving a brawny, doughy sensation. A true edema with complete loss of elasticity of the skin is never present. In addition to this thickening of the subcutaneous tissue, in certain places the entire skin appears to be abnormally loosened upon the underlying tissues. Disturbance of the nutrition of the epithelial elements of the skin gives rise to roughness ,cracking of the nails, coarseness, brittleness and loss of hair, and diminished secretion of perspiration leading to a characteristic dryness of the skin.

Similar changes are seen in the mucous membrane of the nose, pharynx, middle ear, larynx, eyelids, and digestive tract, the changes leading to various functional disturbances, such as mouth breathing, snoring, grunting, a coarse voice, disturbances of hearing, refusal of food, and constipation.

FIG. 245

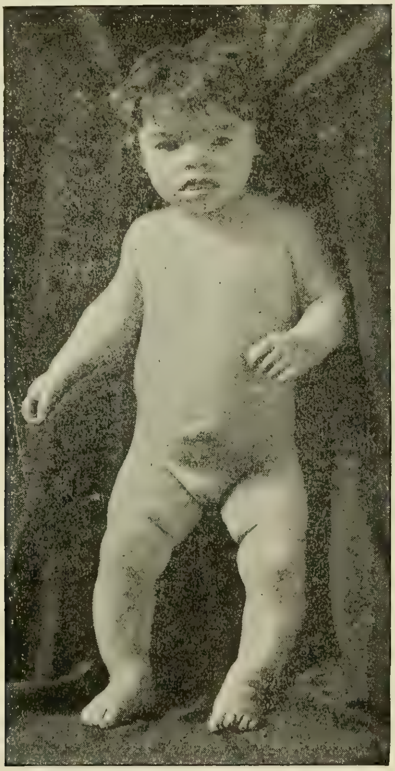


Sporadic cretinism in an infant 10 months old, terminating in sudden death

The changes in the skin and mucous membranes combined with those of the bones and with lack of mental development, give to the cretin its characteristic appearances. The forehead is low and receding, with sparse hair; the nose is broad and saddle-shaped; the opening of the eyes is narrowed by the thickened eyelids; the thickened, protruding lips leave the mouth half open and disclose the thickened and protruding tongue; there is usually a double chim; the ears are large and shapeless; the normal skeletal outlines of the rest of the body are partly concealed by the thickening of the soft parts; there are often pads of thickened skin above the clavicles and over the shoulder blades, hips, and breast; the limbs are cylindrical or columnar in appearance; the hands, fingers, and toes are short and plump; the abdomen is distended and usually shows an umbilical hernia.

THE MENTAL CONDITION.—The mental development not only remains very backward, but even shows a tendency toward deterioration. In the severest cases the intellectual level is little better than that of animals; in average cases it is always that of a very much younger child, and untreated cretins may enter late childhood or adult life with the mental development of a child of two or three years. The characteristics of the cretin are those of apathy rather than of excitability. They are apt to lie all day without showing much reaction to their surroundings, staring apathetically, making automatically only the movements of necessity, or sleeping. When hungry, or in pain, they bawl loudly. Speech may be impossible, or there may be a late acquirement of the ability to say a few words. In less severe cases, the mental impairment shows itself chiefly in inability to fix the attention, to understand, or to talk. They acquire control of the rectum and bladder either very late or not at all. The functions of sitting, standing, and walking are also learned very late.

FIG. 246



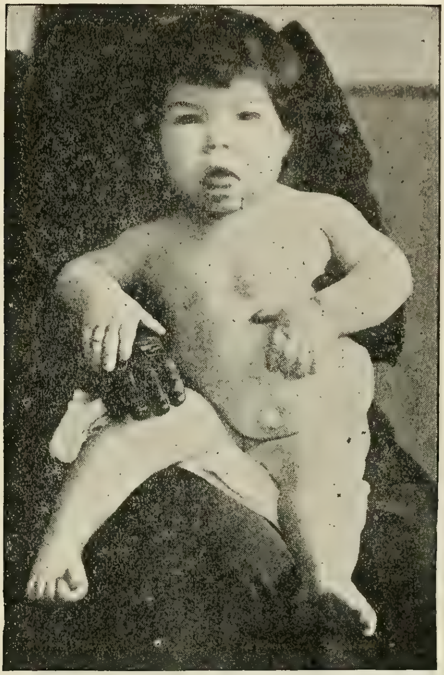
Myxedema. Female, 5½ years old

SEXUAL DEVELOPMENT.—There is usually hypoplasia of the testicles and of the external genitals. Puberty is delayed, and there are various disturbances of the sexual function.

METABOLISM.—Cretins live upon a low plane of metabolism, that of protein, water, and salts being particularly diminished. It is in consequence of this that cretins usually have subnormal temperatures.

MUSCLES.—The musculature is weak and relaxed, although the muscles are not notably diminished in volume. Their condition is closely akin to that seen in pseudomuscular hypertrophy. It is probable that not only the peculiar waddling gait of the cretin is due to muscular weakness but, to some extent also the delay in standing and walking, the tendency toward lordosis, the prominence of the abdomen, and the severe constipation which is so often seen in this disease.

FIG. 247



Myxedema. Female, 9 years old. Slight improvement after eighty days' treatment with thyroid extract.

DIAGNOSIS.—The diagnosis of sporadic cretinism, when the condition is fully developed, is rarely difficult. The appearance of a cretin is so characteristic that the nature of the condition can usually be recognized at once. There is sometimes more difficulty in recognizing the disease in its earlier stages before the characteristic appearance has become pronounced. The physician must be prepared to exclude other conditions in infancy characterized by deficient mental development. He must further be prepared to exclude certain conditions other than cretinism, in which the growth of the bones is affected. The principal conditions to be considered in differential diagnosis are simple backward development, idiocy from organic brain disease, Mongolian idiocy, chondrodystrophy, and rickets.

Simple Backward Development.—In this condition the infant presents none of the peculiarities of appearance characteristic of the cretin. The limbs are of normal length in proportion to the body. There are no abnormalities of the bones, skin, and mucous membranes. The condition is simply that of an otherwise normal child of a younger age.

Congenital Idiocy.—The very common condition of idiocy or feeble-mindedness due to some gross developmental or acquired lesion of the brain, is sometimes mistaken for sporadic cretinism. The only feature which the two conditions have in common is the impaired mentality. Most of these idiotic or feeble-minded children are of an active, restless type, often in constant motion, and do not show the dull placidity of the cretin. Furthermore, their limbs show a normal length in proportion to the body, and their bones, skin, and mucous membranes do not show any of the characteristics of congenital athyreosis. As an additional point in the diagnosis between the two conditions, in cases of congenital idiocy there are often some stigmata pointing directly toward the lesion of the central nervous system. The commonest of these stigmata is tonic spasm of the extremities, but disturbances of the reflexes, paralysis, blindness, and so forth, are often seen. In differentiating between the two conditions, the physician should examine the bones, skin, and mucous membranes for all the signs of cretinism, should measure the length of the limbs in proportion to the body, and should look carefully for all the signs of a lesion of the central nervous system.

Mongolian Idiocy.—This is the condition which presents most difficulty in the differential diagnosis from sporadic cretinism. The distinction between the two conditions is plain after the condition of cretinism is fully developed, but in the early months of infancy, with an infant of backward development and a peculiar facial appearance, it is often difficult to say whether we have to do with a mongol or a cretin. The characteristic signs of mongolism are the mongol slant to the eyes, the broad, flat bridge of the nose, and the over-development of the epicanthic folds of the eyes. These are the only stigmata of Mongolian idiocy, but they are also seen at times in sporadic cretinism, particularly when the infants are poorly nourished. The most important evidence in differential diagnosis is to be gained from measurements of the proportion of the length of the limbs to that of the entire body, which is always diminished in the cretin, but never diminished in the mongolian idiot. Furthermore, the myxedematous changes in the skin and mucous membranes are not seen in the mongol. The mongol also reacts very vigorously to his surroundings, and is less dull and placid than is the cretin.

Chondrodystrophy.—This condition is sometimes mistaken in early infancy for sporadic cretinism. It is exclusively a disease of the bones, and shows no abnormality of mental development, or of the skin and mucous membranes. Infants with chondrodystrophy entirely lack the cretinoid appearance. However, the failure of these children to gain in length, with their short limbs and short pudgy hands, is suggestive of cretinism, the physician being in doubt whether it may not be too early for the other cretinoid characteristics to show. In sporadic cretinism, when the process has advanced far enough to cause perceptible shortening of the limbs, the other signs of cretinism should be present. In case of doubt, the Roentgen ray will easily distinguish between the two conditions.

Rickets.—Rickets should not be mistaken for cretinism. All the cretinoid characteristics are lacking. The limbs are not short in proportion to the body, and the nature of the disease is apparent from the specific rachitic changes in the bones, rosary, enlarged epiphyses, and so forth.

PROGNOSIS AND TREATMENT.—As might be inferred from the nature of the disease, there is no tendency toward spontaneous improvement. Untreated cretins may live to an advanced old age, but they remain dwarfs and idiots, and retain the physical characteristics of the disease. The therapeutic indication is to supply the lacking thyroid secretion. The simplest and most rational method of attaining this end would seem to be the transplantation of thyroid tissue into some part of the body of the patient. This has been tried many times, but although the transplanted tissue appears to develop in the body of its new host, it does not appear to give a long continuance of normal function, which is probably to be explained upon the ground of the biological law of individual specificity. Fortunately we can supply thyroid secretion, which is apparently thermostabile, by giving a preparation made from the thyroid gland of an animal. Various preparations have been given in various ways, but the one most commonly employed is the dried powdered gland usually called *thyroid extract*, which is given by mouth. Most of the thyroid extracts on the market are prepared from the glands of the sheep. It is essential that a reliable extract should be used. It is best to begin with doses of half a grain once or twice a day, and when the patient has become accustomed to it, the doses may be gradually increased until the child is taking from 3 to 5 grains daily. Occasionally at the beginning of treatment, some disturbance is caused by the administration of thyroid extract, particularly irritability, marked perspiration, rise of temperature, and an increase in the pulse rate. These symptoms soon pass off, however. For long standing cases in older children, at least 5 grains daily should be given.

The improvement after the use of thyroid extract is very remarkable. After a few months, or even after a few weeks of treatment, the entire appearance of the child is changed. The abnormalities of the skin and mucous membranes, and the idiotic expression of the child quickly disappear. There is a marked increase in growth in length, muscular power develops rapidly, and progress is made in dentition. The child usually learns to walk, and there is an improvement in the mental condition, so that he begins to take a more and more active and intelligent interest in his surroundings. It must be remembered, however, that intellectual progress is much slower than the purely physical improvement. If treatment is begun early, all the physical characteristics of the cretin may disappear. In the meantime, the children become much brighter and learn to talk. A certain number of cases become so much improved intellectually that they are able to attend school. Nevertheless, the mental development never becomes wholly normal, and parents should not be led to expect as much as this. Even when children are intelligent enough to go to school, they are always much behind other children of the same age, both in mental and in physical development, and while they may live active, happy and useful lives in certain occupations, they never become wholly normal. It is essential that the administration of the thyroid extract be continued throughout life. It if be omitted even for a few weeks, deterioration begins at once, and a relapse is seen.

HYPOTHYROIDISM

(Late Infantile Myxedema)

This name is given for convenience to a condition in which there is not a complete congenital absence of thyroid tissue, and in which the symptoms are due, not to an absence, but to a deficiency of thyroid secretion.

ETIOLOGY.—A certain number of these cases should upon an etiological basis be classified with sporadic cretinism, because they represent a congenital malformation which differs only from that described above in that there is not complete absence of thyroid tissue, but only an imperfectly formed gland which is functionally insufficient. The majority of these cases, however, belong in the class of acquired myxedema, and are due to an injury to the thyroid gland which takes place during extrauterine life. Such cases represent much the same condition as is seen in the myxedema of adults. When the injury which produces the lesion of the thyroid is something peculiar to a definite locality, the condition is called *endemic cretinism.* This form is seen in certain parts of Switzerland, Italy, and France, and in certain districts near the great lakes of America. There is some evidence that the injurious agent is conveyed in the drinking water. Other cases of acquired myxedema are wholly sporadic and are to be traced to various acute diseases in which the thyroid gland is involved.

PATHOLOGICAL ANATOMY.—The condition in the gland is sometimes a primary atrophy, and sometimes a goiter-like degeneration with more or less complete disappearance of the glandular tissue. Various forms of thyroid degeneration are seen in the endemic cases.

SYMPTOMS.—The symptoms are generally the same as those which have been described under sporadic cretinism, differing chiefly in their severity and in the time of their appearance. Every variation is seen from children who show very little deviation from the normal to children who show a condition closely approaching that described under sporadic cretinism. In the majority of these cases symptoms are not noted until the second or third year, or even later. Usually the first thing which attracts attention is a failure of mental or physical development. It is noted that the child is backward, is unable to learn what is taught, pays no attention to commands, seems unable to fix its attention, and is not cleanly in its habits. In other cases it is noticed that it is small for its age, some children being greatly stunted. Measurements of the limbs may or may not show shortening of the limbs in proportion to the body. Roentgenograms usually show a backward ossification, but there are usually some carpal, tarsal and epiphyseal centers of ossification, indicating that there has been at least some thyroid activity. The facial expression varies from that characteristic of the cretin to one that is simply expressionless or rather stupid. There is a variable amount of thickening of the skin, lips, tongue, and eyelids. The hair may or may not be coarse and thick. The abdomen is usually prominent, and often shows an umbilical hernia. The voice is often deep and hoarse, and dentition, both the first and the second, is delayed and irregular. Constipation is very marked.

DIAGNOSIS.—The ease with which these conditions of hypothyroidism may be recognized varies greatly with the individual case. In marked cases with a notable deficiency of mentality and obvious physical characteristics of the cretin, the diagnosis of late cretinism is not difficult. In less marked cases the physician should remember the possibility of hypothyroidism and should apply the therapeutic test. Rapid improvement under thyroid extract is a sign that the diagnosis is correct.

PROGNOSIS.—Marked improvement occurs under thyroid extract, but as in sporadic cretinism, it is a doubtful question in any case whether a normal condition will ever be gained.

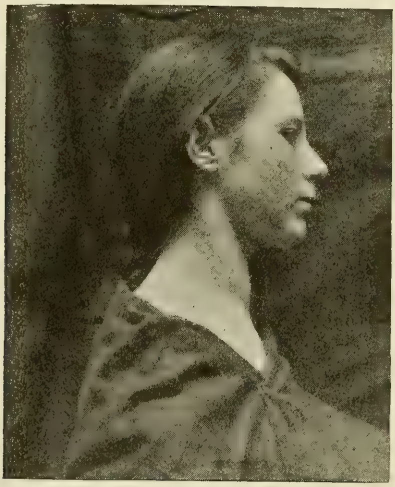
TREATMENT.—Treatment is the same as that of sporadic cretinism.

GOITER

(Bronchocele)

Enlargement of the thyroid gland is commonly called goiter. True goiter consists in the enlargement of the old and in the formation of new alveoli, in the cells of which a greater or less amount of colloid degeneration takes place. The colloid abnormalities of goiter are rarely present in children, in whom the thyroid enlargement seems to be little more than a continuation of natural growth and a true hypertrophy or an excessive development of normal tissue. Usually the enlargement of the gland is the only symptom. Infants have been born with an enlarged thyroid.

FIG. 248



Hyperemia of the thyroid gland. Female, 13 years old

The disease is endemic in Switzerland, in certain parts of France and Italy, and in Michigan. The enlargement may be purely vascular (hyperemia of the thyroid), parenchymatous, or cystic. In places in which the disease is endemic the cause in many cases seems to be connected with the drinking-water. It has a certain relation to the endemic form of myxedema, the nature of which has not been fully determined. A woman with goiter may, on removal to a place in which the disease is endemic, give birth to a cretin. Although a simple goiter generally gives rise to no symptoms other than those of pressure, symptoms of myxedema may develop. The inference is that this complication is due to interference with the function of the gland, as a result of which the secretions are either diminished or entirely checked. When myxedema does not develop, as it does not in the majority of cases of goiter, we may account for the fact on the supposition that the enlargement of the gland has not affected its function as a secreting organ.

HYPEREMIA OF THE THYROID.—Between the ages of twelve and fifteen years in girls, about the time of puberty, an active hyperemia of the rich vascular tissue of the thyroid occurs, leading at times to a considerable and rapid enlargement of the gland. Anemia, especially chlorosis, palpitation, accelerated pulse, and various nervous symptoms are not uncommon at this period, and when noted in connection with the hyperemic enlargement of the thyroid, may give rise to the belief that the condition is the beginning of the much more serious disease exophthalmic goiter. This enlargement of the thyroid does not usually persist, however, the disease generally subsiding under appropriate treatment of the anemia, and the diagnosis is readily made.

EXOPHTHALMIC GOITER

(Grave's Disease)

Exophthalmic goiter occasionally, but rarely, occurs in childhood, independently of the physiological disturbance which has been described under hyperemia of the thyroid gland.

ETIOLOGY.—According to Sachs, heredity plays a much more important part in children in the production of the disease than the emotional excitement, fright, cardiac disease, and severe constitutional disorders which so frequently underlie the condition in adults. Epilepsy, chorea, and chronic alcoholism in the parents have been said to predispose to the development of the disease in children.

The most recent views attribute the disease to hypersecretion of the thyroid gland, to which the name *hyperthyrea* is given, as opposed to the *athyrea* of myxedema.

SYMPTOMS.—In some cases the onset is acute, in others subacute or chronic. The disease is characterized by the cardinal symptoms of tachycardia, exophthalmos, muscular tremors and enlargement of the thyroid, and by general symptoms of anemia, indigestion with a special tendency to profuse diarrhea, slight fever, loss of weight, and many other symptoms of a neurasthenic character.

Tachycardia.—The pulse rate is increased to 90, 100, 120, or even 200 per minute, the rate being increased in states of excitement and lowered when mental and physical rest is enforced. There is no evidence of an organic lesion in the heart. All the arteries throughout the body pulsate with unusual distinctness. Owing to the increased blood-tension, hemorrhages may occur from the nose, stomach, or intestines. Upon palpation of the goiter a distinct thrill may be felt.

Exophthalmos.—The protrusion of the eyeball is not accompanied by disturbance of vision. Limitation of the field of vision with ulceration of the cornea from want of protection of the lids may occur. Graefe's symptom, a failure of the upper lid to follow promptly a downward movement of the bulb, Stellwag's symptom of a dilated palpebral fissure, and Moebius's symptom of defective convergence of the axes of the two eyes are signs to be noted in connection with the exophthalmos, but are not always present.

Thyroid Enlargement.—The thyroid is almost always enlarged, but the increased size may follow rather than precede the other cardinal symptoms. The enlargement is usually bilateral and symmetrical, the tissues are vascular, hyperplastic, and may show fibrous degeneration.

Muscular Tremors.—The muscular tremors are usually rhythmical, and at the rate of about eight to the second.

Throbbing of the blood vessels and free perspiration are unpleasant symptoms, adding to the discomfort and nervousness of the patient. Intense pigmentation of the skin, resembling Addison's disease, may rarely occur, or areas of leukoderma, or of urticaria. Very rarely myxedema has developed towards the end of the disease.

DIAGNOSIS.—The diagnosis is made by the recognition of the cardinal symptoms. Physiological hyperemia of the thyroid at puberty should not be diagnosed as exophthalmic goiter.

PROGNOSIS.—The course is generally chronic and of several years' duration. Some cases recover, but if the disease is once well developed it is apt to be prolonged, death often ensuing from some intercurrent affection.

TREATMENT.—Absolute rest in bed, avoidance of all excitement, and careful regulation of the diet are essential. Digitalis and strophanthus are sometimes useful. Good results have been reported from the use of belladonna or of atropin. The galvanic current is also recommended. The thyroid extract has proved of no value in these cases. When the gland is so large as to cause symptoms of pressure, partial extirpation is to be considered. The operation has been done in adults with good results in a certain number of cases.

Recently subcutaneous injection of thyroidectomized goat lymph has been tried, and in some cases with success.

ACUTE THYROIDITIS

Acute inflammation of the thyroid is not very common, but may occur from a variety of causes. It may result in the formation of abscesses of various sizes or in the production of new connective tissue. Acute thyroiditis is rarely primary, being commonly a metastatic affection occurring in the course of some febrile disorder. It has been noticed among children as a complication of measles, typhoid fever, diphtheria, and parotitis, and the process in a majority of these recorded cases, instead of retrograding spontaneously as it did in others, caused an inflammatory condition in which abscess-formation occurred. On opening the abscesses the pus was found to contain numerous micrococci.

SYMPTOMS.—The symptoms of acute thyroiditis are swelling, redness, and tenderness of the gland. Symptoms of pressure, such as dyspnea, hoarseness, painful deglutition, and neuralgic pains, are sometimes present with fever and general malaise. The duration of the disease is from two to three weeks.

TREATMENT.—The treatment is essentially expectant, but some previously intractable cases seem to have been benefited by the application of iodin. The patient should be carefully watched, and if there are indications that suppuration has taken place, an incision should be made at once, as recovery then usually occurs quite quickly.

ENLARGED THYMUS

(Status Lymphaticus; Thymic Asthma)

This is a condition having a very definite pathological anatomy, but in which the clinical manifestations are neither constant nor characteristic, and bear a relation to the lesions which is not clearly understood. The reason that the pathogenesis of the various symptoms which may accompany enlargement of the thymus is not clear, is that little is known of the physiological function of the gland, and consequently no clear relation between the symptoms and the functional disturbance produced by the lesion can be observed.

PATHOLOGY. STATUS LYMPHATICUS.—Attention was first attracted to enlargement of the thymus by the occasional occurrence of sudden death, without any recognized pathological process being found as an adequate cause. In these cases post-mortem examination often disclosed a condition of marked hyperplasia of the thymus, associated with a hyperplasia of the lymphoid tissue throughout the body. This involves the lymphnode groups, the tonsils, the follicles at the base of the tongue, the lymphoid follicles of the intestine, and the follicles of the spleen. Paltauf first came to the conclusion that this condition represents an abnormal constitutional state, and gave it the name of *status lymphaticus*. Other characteristics of status lymphaticus as thus described are a hypoplasia of the circulatory apparatus, a narrowing and thinning of the walls of the aorta and smaller arteries, and, in older individuals, a blood picture like that of chlorosis.

ENLARGED THYMUS WITHOUT OTHER SIGNS OF STATUS LYMPHATICUS.—Whether or not we accept Paltauf's theory of status lymphaticus as a clinical entity, we must admit that there are many cases in which hyperplasia of the thymus is the sole lesion, all the concomitant signs of status lymphaticus being absent. It is possible that the majority of instances of enlarged thymus belong in this class. Whether or not these cases are just as liable to sudden death as typical cases of status lymphaticus is not finally determined. It is known, however, that there are certain symptoms which, while they may be seen in typical status lymphaticus, may also be produced by hyperplasia of the thymus alone. These symptoms cannot be attributed to a general constitutional abnormality, and their origin must be sought in the particular lesion of the thymus. This fact increases the difficulties in giving a clear clinical description.

Many careful statistical observations have been made upon the normal weight of the thymus in infancy and early childhood. The weight is greatest at birth, averaging about 7 grams. Between birth and five years, the average normal weight is about 4 grams. Any weight over 10 grams should be considered abnormal. In cases of enlarged thymus, the weight may be from 15 to 50 grams; I have seen one thymus weighing 60 grams. In cases of moderate enlargement the weight is 15 or 20 grams, and in marked enlargement 30 or 40 grams. There are no characteristic gross or histological changes other than hyperplasia.

Various theories have been advanced in explanation of the symptoms which are associated both with status lymphaticus and with simple enlargement of the thymus. The inadequacy of all explanations is shown by the fact that the lesions need not necessarily produce any symptoms whatever. Many cases are reported in which sudden death in apparently healthy children is attributed to the thymic enlargement found at autopsy. There can be no doubt that some of the symptoms observed are due to *pressure*, either upon the trachea, or upon the great vessels. A mechanical explanation, however, does not account for all the clinical phenomena observed at times in these cases. It does not explain the tendency toward sudden death, and the lessened resistance to intercurrent infections. These phenomena have been explained under the pressure theory on the basis of a sudden engorgement of the thymus with marked increase in pressure, but the explanation is not wholly satisfactory. Furthermore, even in cases in which the most striking clinical phenomenon is respiratory disturbance, pressure, while undoubtedly often playing an important part, does not offer a full explanation. In most cases the dyspnea is paroxysmal rather than continuous, and stridor is not marked in proportion to the severity of the dyspnea. The following are the principal theories besides that of pressure which have been advanced in explanation of the symptoms associated with enlargement of the thymus: (1) *Hyperthymization;* (2) *intoxication from faulty tissue change;* (3) *intensified anaphylaxis.*

None of these theories has much evidence in its support. They will be further considered under Problems and Research.

SYMPTOMS.—As stated above, the enlargement of the thymus may cause no symptoms. In infancy, status lymphaticus is one of the causes of sudden death from slight apparent cause, and there may be nothing to indicate the existence of the condition until this occurs. The sudden fatal ending may be brought on by such causes as a fall or other accident, anesthesia, a surgical operation, or some acute disease. Death may be preceded by repeated convulsions, high fever, or paroxysms of marked dyspnea with or without signs of mechanical obstruction to the respiration. In most cases of this type the general signs of status lymphaticus are found in addition to the enlargement of the thymus. In some cases tracheal obstruction, or pressure on the circulatory or nervous systems, is the probable cause of death, but we do not know to what extent some form of general intoxication plays a part in these cases.

In many other cases of enlarged thymus, the first attack does not result in death, or there is never a fatal attack, and it is these cases which are of the greatest clinical interest. The most important and characteristic symptom is *dyspnea*, and it is for this reason that the name *thymic asthma* has been applied to the condition.

The dyspnea may be continuous or intermittent. The continuous form is seen most often in very young infants. The respiration is increased in rate, and is more or less labored. At times there is an increase in the respiratory difficulty, which culminates in a suffocative attack accompanied by intense cyanosis. These attacks are repeated at varying intervals, there being always some dyspnea between attacks. In this form, with continuous dyspnea, there is usually also *stridor*. The stridor may or may not be marked during the intervals between attacks, but is usually intense during the paroxysm. It is inspiratory in character.

The intermittent form is more common in my experience. It is seen usually in children who are at least several months old, and is the type seen more often in the latter half of the first year, and sometimes in the second year. It is characterized by paroxysms of dyspnea in children who between attacks are apparently quite normal. I have seen two clinical varieties of this intermittent form. In the first there is a sudden suffocative attack, accompanied by cyanosis and inspiratory stridor, lasting only a few moments. This variety may be explained upon purely mechanical grounds. In the second variety the paroxysm lasts longer, but is not so intense. The breathing in this variety is extremely rapid, but is less labored, there is little or no cyanosis, and suffocation is not threatened. In many cases of this variety, stridor is entirely lacking, the attack being a paroxysm of extremely rapid breathing which is often not markedly labored, without any signs suggesting mechanical obstruction. In other cases of this type the dyspnea may be expiratory, with asthmatic râles, or there may be both inspiratory and expiratory dyspnea. The duration of the attacks in this variety varies from a few hours to several days.

The explanation of the symptoms in these two forms of the disease is not wholly clear. In the continuous form, with suffocative attacks and marked inspiratory stridor, there seems little doubt that the chief cause is mechanical obstruction to the respiration. The same is true of the cases of the intermittent form in which inspiratory stridor is marked, the breathing very labored, and cyanosis present. The obstruction may come from pressure of the thymus on the trachea, or from an associated laryngeal spasm. Not all cases of congenital stridor are due to enlarged thymus, as there is a congenital laryngeal stridor due to a peculiar formation of the larynx.

In many cases of the intermittent type, the mechanical explanation of the symptoms does not seem very satisfactory. The intermittent character of the symptoms might be explained by periods of thymic engorgement and increased pressure, but there is no proof of such an explanation. The cases without stridor or with only expiratory dyspnea, without cyanosis, and with paroxysms of rapid breathing which does not suggest obstruction, are very strongly suggestive of a factor besides the purely mechanical one, namely, some form of *intoxication*.

There are few symptoms of importance other than the respiratory. Occasionally dysphagia is present. Occasionally there are signs of pressure on the great vessels.

DIAGNOSIS.—The diagnosis of enlargement of the thymus depends upon (1) physical examination; (2) X-ray examination; (3) certain special methods of examination.

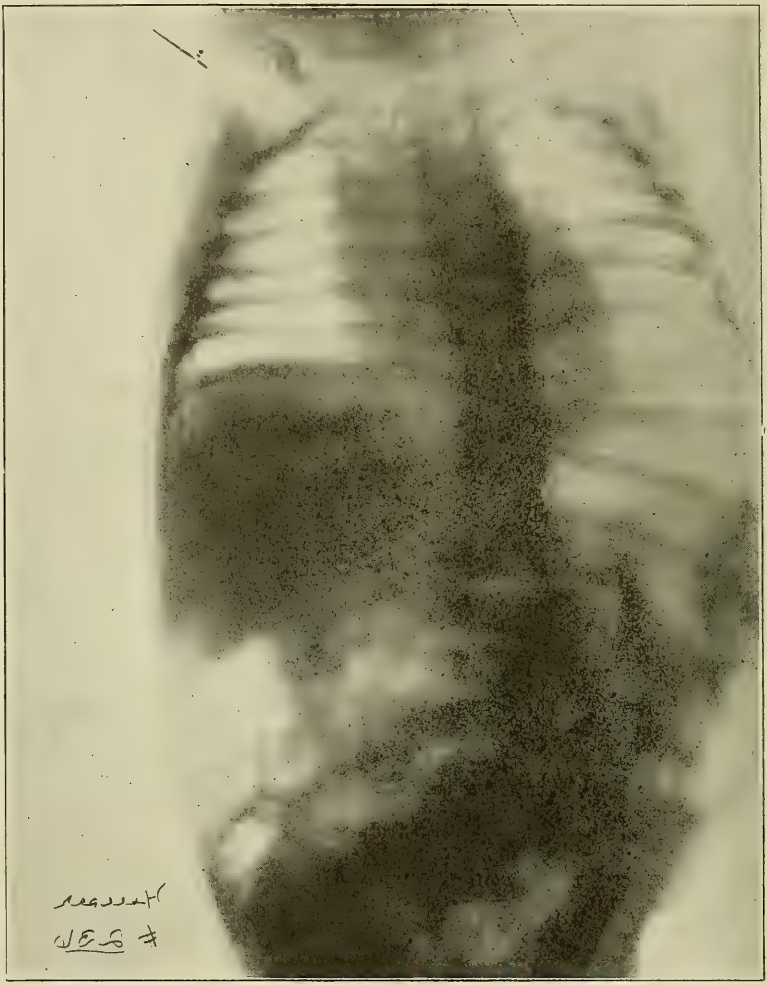
PHYSICAL SIGNS.—The most important evidence on physical examination is to be gained from percussion. In infants there is a slight but evident area of normal thymic dulness in the shape of an irregular triangle or truncated cone, of which the base is at the sternoclavicular junction, and the apex at the second rib. This dulness can only be detected on very light percussion, or with special methods of percussion. It extends laterally very little beyond the margin of the sternum, slightly more on the left than on the right. An increase in the quality of this normal dulness under the manubrium, or dulness extending more than 1 cm. beyond the sternal margin on either side is, in the absence of other causes, suggestive of thymic enlargement. When the dulness is continuous with the cardiac dulness, concealing the note of pulmonary resonance between the heart dulness and the normal area of thymic dulness, it is further evidence of enlargement of the thymus. Enlarged or caseous lymphnodes at the hilus of the lung do not give dulness in this area Simply engorged lymphnodes in the anterior mediastinum also do not produce dulness in this area, but caseous lymphnodes in this region do. Another sign of enlarged thymus only seen occasionally is a palpable mass or visible expiratory bulging in the region of the jugulum.

X-RAY EXAMINATION.—The diagnosis of enlarged thymus has been much simplified by the use of the X-ray. The shadow of the normal thymus occupies a V-shaped area which scarcely extends beyond the limits of the median bony structures. An enlarged thymus gives a wide median shadow above the heart, appearing like a broad cap superimposed on the shadow of the heart and great vessels. When the shadow in this region extends more than ½ or ¾ cm. beyond the bony outlines on either side, it is suggestive of thymic enlargement. Normally there is no shadow outside the bony structures down to the point where the regular outlines of the heart begin to appear. A shadow above this point and outside the bony outlines continuous with the heart shadow, is seen in thymic enlargement.

Broadening of the shadow in this region is not always due to enlarged thymus. At times, in infants, a broadening of the shadow to the right may be due to the great vessels, but notable broadening to the left is only due to the thymus. The shadow of enlarged bronchial lymphnodes may sometimes suggest a thymic enlargement. Usually, however, enlarged bronchial lymphnodes give an extra median shadow to one side, which does not connect with the heart shadow.

SPECIAL METHODS OF EXAMINATION.—Various special methods of percussing the thymus, and even special instruments have been devised. These, however, are not adapted to use in ordinary practice. Skilled observers have been able to demonstrate the existence of tracheal stenosis by means of the bronchoscope. This also is not of much practical value. In some cases of thymic enlargement a notable lymphocytosis has been found on blood examination. This would be of more value in older children than in infants, but cannot be taken as more than a confirmatory sign.

FIG. 249



X-ray of enlarged thymus

DIFFERENTIAL DIAGNOSIS.—Enlarged thymus must be differentiated chiefly from the other causes of dyspnea and obstructed breathing. The other causes of stridulous dyspnea are enlargement of the bronchial lymphnodes, retropharyngeal abscess, membranous laryngitis, catarrhal spasm of the larynx, laryngospasm, congenital laryngeal stridor, and asthma. Enlarged thymus must also be differentiated from the diseases of the heart and lungs which are associated with dyspnea.

PROGNOSIS.—The outlook in any case in which a diagnosis of enlargement of the thymus has been made is very uncertain. While any attack, no matter how severe the symptoms, may be recovered from, it must always be remembered that these cases are likely to have a low resistance to injurious influences, and are liable to sudden death from comparatively slight cause.

TREATMENT.—Cases of thymic enlargement showing any symptoms should be treated by means of exposure to the Roentgen rays. The influence of this process upon the lymphoid structures in general, and upon the thymus in particular, has been proved beyond the possibility of doubt, and it must be regarded as an established fact that X-ray exposures are capable of inducing involution of the thymus gland.

When the symptoms are urgent, especially with evidence of marked mechanical obstruction, the exposure should be given daily for four or five days, or until a marked improvement has been obtained. It has been shown in rabbits that four exposures on successive days gave a greater degree of fibrosis than fifteen exposures over five and a half weeks. When the symptoms are not urgent, or after a notable improvement has been obtained, the exposures may be given at intervals of a week. The time of exposure should be five minutes. The results obtained even from a single exposure, are often very striking. It has been noted that symptoms which gradually disappear under X-ray treatment sometimes tend to recur, disappearing again when the treatment is resumed. This is to be explained by partial regeneration in the thymus, and shows that it is possible to regulate the treatment according to the necessities of the case with little danger of complete loss of thymic function.

Surgical intervention was the first treatment sought in enlarged thymus. The first operation was elevating the thymus and fastening it to the tissues of the neck or the top of the sternum. The results were not good, and it soon gave place to thymectomy. The mortality of this operation has been high. Furthermore, it seems probable in the light of recent research, that the complete removal of the thymus during the period of its functional activity is likely to be fraught with grave danger to the development of the child. The success of X-ray therapy is great enough to render surgical intervention entirely unnecessary.

PROBLEMS AND RESEARCH.—The literature of enlarged thymus has been occupied mainly with theoretical considerations. The theories of the cause of sudden death in status lymphaticus, and of the symptoms of hyperplasia of the gland, have been much discussed. There is no doubt that mechanical obstruction is a possible cause both of sudden death and of the respiratory symptoms. The theories that the clinical phenomena are due to hyperthymization or to intoxication from the products of faulty metabolism, are without much supporting evidence. The view that the symptoms of status lymphaticus represent an anaphylactic phenomenon is ingenious. It is based on the observation of intensified forms of such diseases as pneumonia and tuberculosis. In patients with status lymphaticus, it is argued without proof that fulminant types of illness are examples of anaphylaxis, and that the status lymphaticus intensifies anaphylactic phenomena.

Of positive accomplishment in the investigation of the thymus, there is very little. The changes which take place in thymectomized dogs have been thoroughly studied and described. It may be regarded as established that the thymus is a necessary organ in infancy, and there is strong evidence of an internal secretion, based on the results of thymectomy. Such evidence has to do with an association with the chromaffin system, with a relation to other ductless glands, and with an influence on nutrition in general. On the other hand, all attempts to produce hyperthymization by feeding have failed. Experimental research on the results of the feeding of thymic extract has failed to give definite evidence of an internal secretion. Finally, there is a question as to the possible relation of the thymus to such conditions as cretinism, mongolism, and chondrodystrophy.

ADDISON'S DISEASE

ETIOLOGY.—Addison's disease is even more rare in children than in adults, only about twenty cases having been reported. The cause of the disease is still an open question. The same symptoms occur when different pathological conditions are present. The adrenal glands are supposed to furnish an internal secretion which is necessary for normal metabolism, and a loss of this secretion is considered by some writers to give rise to the symptoms, while others believe that the disease is dependent upon changes in the ganglia of the sympathetic nervous system of the abdomen.

PATHOLOGICAL ANATOMY.—The pigmentation varies from a light yellow to a deep bronze. It is usually diffuse, and is most intense on the exposed parts of the body, such as the head and hands, and in the flexures, around the nipples and about the genitals. The mucous membranes of the mouth and vagina are likewise pigmented, but the palms and the soles remain free for a long time. Small areas of leukoderma may appear.

SYMPTOMS.—The onset is usually insidious. Progressive loss in strength, and a characteristic discoloration of the skin are the conspicuous symptoms of the disease.

Vomiting and diarrhea are especially common when the disease occurs in children. Nervous symptoms are sometimes marked, and are of the same character as those we see in cases of severe secondary anemia. The blood, however, shows only a slight degree of anemia. There is no emaciation except in the later stages.

The course of the disease is progressive, with occasional remissions, the duration varying from a few months to one or two years. Death occurs from exhaustion or from the development of an intercurrent affection, which may appear as some form of tubercular disease in other parts of the body.

On autopsy the adrenal glands are frequently found to be tubercular, or the seat of other lesions.

DIAGNOSIS.—In the diagnosis of Addison's disease other causes of pigmentation of the skin must be excluded, such as may arise from arsenic, lead, nitrate of silver, malaria, and abdominal growths. The tuberculin test is often of value as an aid in the diagnosis when the cause is due to tuberculosis of the adrenal glands.

PROGNOSIS.—The disease is nearly always fatal. A few recoveries have been reported.

TREATMENT.—The adrenal glands of the sheep have of late been used frequently in the treatment, and in a few cases with improvement in the symptoms. The remedy is worthy of a trial, but the results are usually very disappointing. The glands may be given raw or slightly cooked, in the form of a glycerin extract, or as a dry extract in tablets. From one-half to one gland may be given daily, or one-grain doses of the dried extract three times a day. General treatment and measures directed to the control of the symptoms are indicated.

DISEASES OF OTHER DUCTLESS GLANDS

It is a tendency in modern medicine, possibly an unfortunate one, to ascribe a great variety of functional disturbances of obscure pathogenesis to a disease of some of the ductless glands. This is because there is so much in connection with the physiology of the glands having internal secretion, which is not yet understood. Acromegaly, which in adults is due to a lesion of the pituitary gland, is practically never seen in early life. There is a condition rarely seen in children associated with tumors of this gland, which has been described as Fröhlich's syndrome. The symptoms are adiposity, delayed sexual development, and sometimes mental dulness. Cases have been reported in which precocious sexual development was attributed to tumors of the pineal gland, or to tumors of the adrenals.

The indiscriminate prescribing of glandular extracts for obscure conditions in early life cannot be too strongly condemned. Enormous additions to our knowledge are required before we can ascribe certain conditions of infantilism, and of disturbance of various functions, definitely enough to the ductless glands to afford a basis for rational therapy. In the meantime it is quite possible that the indiscriminate use of glandular extracts may be fraught with considerable danger.

DIVISION XIV

DISEASES OF THE BONES, JOINTS AND MUSCLES

OSTEOGENESIS IMPERFECTA

(Fragilitas Ossium; Osteopsathyrosis)

This is a disease characterized by excessive fragility of the bones, for which no definite cause can be found. It is a fetal disease, the greater number of cases being still-born. Some cases survive, and present a very characteristic clinical picture. Occasionally this condition of fragility of the bones, instead of being manifest in infancy, does not appear until later childhood. These cases were formerly spoken of as *idiopathic osteopsathyrosis*, but modern opinion regards the condition as the same process of fetal origin as in congenital osteogenesis imperfecta, the symptoms not appearing until after a period of latency.

ETIOLOGY.—The cause of the disease is entirely unknown. Heredity seems to be the only important factor in favor of which there is any evidence, a number of writers having observed several cases in the same family. There is no evidence of any relation to syphilis, or to disease of the ductless glands. The immediate cause seems to be some fault in the condition of the mother during pregnancy.

PATHOLOGICAL ANATOMY.—Macroscopically the bones show multiple, irregularly placed fractures, either recent, or healed with slight callus formation. The bones may be soft and pliable, or very brittle; they may be normal in length, or short and thick. The bones of the skull show an almost complete lack of calcification, the cranium being a soft membranous bag. The cortex of the long bones is extremely thin, and in marked cases the shaft of the bone may consist only of a firm periosteum enclosing a reddish-brown mass intersected by fine bony spicules.

Microscopically, the region between the epiphysis and the diaphysis of the long bones shows no abnormality in the proliferation of the cartilage. The conversion of cartilaginous into bone tissue is markedly reduced in amount, but otherwise takes place normally. This leaves numerous cartilaginous remnants in the diaphyses, and a markedly diminished number of bony trabeculae. The number of osteoblasts

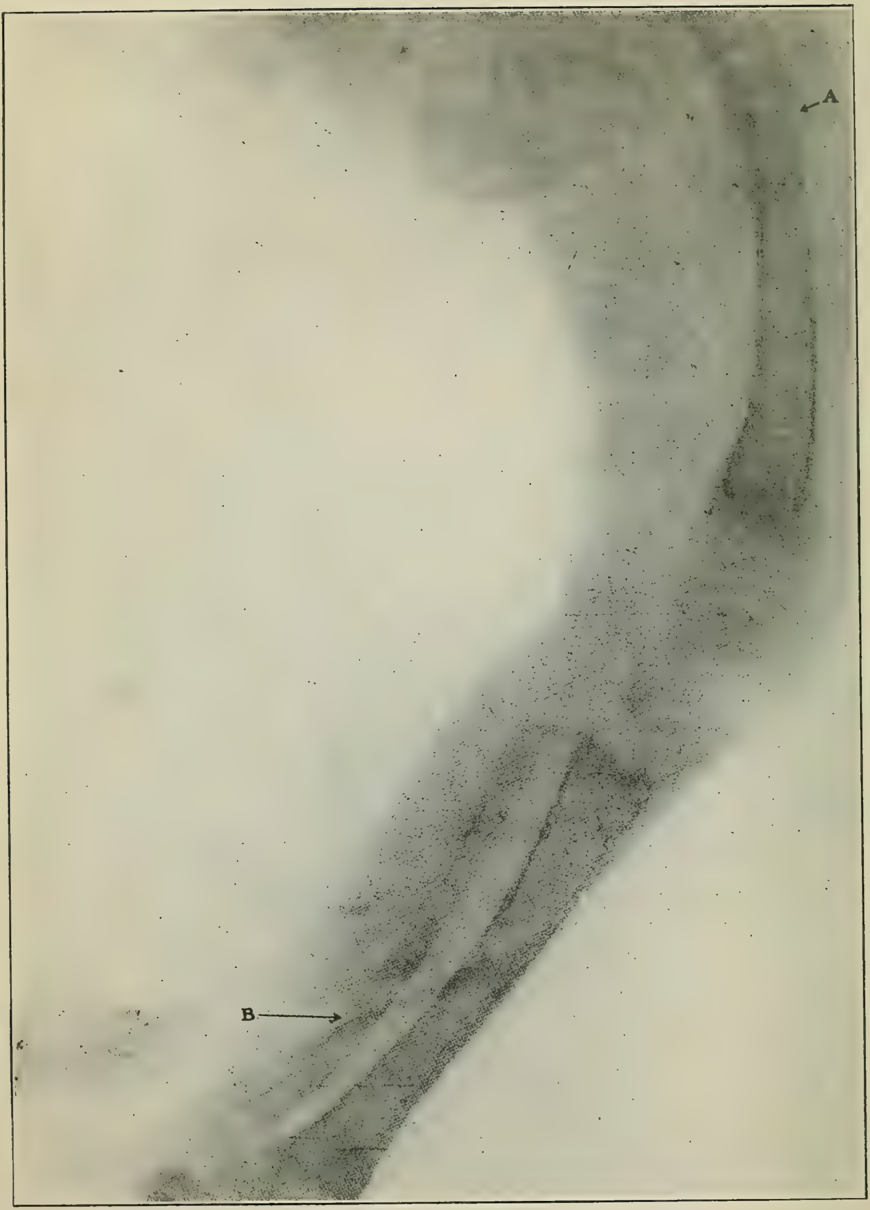


FIG. 250—Osteogenesis imperfecta

is everywhere diminished, and the marrow is markedly increased. Periosteal ossification is absent in some places, slight in others.

SYMPTOMS.—Infants who survive birth often present an easily recognizable picture, on account of the deformities produced by the multiple fractures. The extremities show all sorts of deformities, with shortening and bending. Callus formation is not marked, but a certain amount can be detected. The fontanels and sutures are widely open, and often the entire cranium feels soft and boggy like parchment. The thorax usually shows no deformity except more or less numerous, small knob-like protuberances on the ribs representing healed fractures. In some cases there is such extreme fragility of the bones that the slightest manipulation causes new fractures. In other cases the stage of extreme fragility appears to have been passed through in utero, the chief appearances being malnutrition and deformities.

In addition to the bony abnormalities, infants with osteogenesis imperfecta are usually undersized and poorly nourished, with soft delicate skin and long silky hair. The face is normally formed. The abdomen is protuberant, but without umbilical hernia.

In some cases, now believed to represent a less marked severity of the same disease, the children are apparently normal in infancy, but in childhood, when their physical activity increases, they show a tendency to fracture from very slight violence Fractures in this disease appear to cause little pain, and tend to unite rather rapidly.

DIAGNOSIS.—The diagnosis of osteogenesis imperfecta is usually easy. The only other condition in infancy in which spontaneous fractures are seen is rickets. In rickets, however, the tendency to fracture is never so extreme, and while there may be more than one fracture, the multiple lesions characteristic of osteogenesis imperfecta are not present. Furthermore, rickets is recognized by the characteristic changes at the epiphyses of the long bones, which are absent in osteogenesis imperfecta. The X-ray appearance of the bones in osteogenesis imperfecta shows large medullary cavities with a very thin atrophic cortex The epiphyseal lines are fairly sharply defined.

PROGNOSIS.—The prognosis of the disease is bad. Most of the infants who are not still-born die within a comparatively short time after birth, but those which survive longer have a low resistance, and usually die during infancy from some intercurrent affection. The prognosis of the cases in which the disease first manifests itself in later childhood is better. Some of them have appeared to undergo spontaneous cure. The majority, however, have to live in constant dread of new fractures, and are often more or less incapacitated by the deformities which have been produced.

TREATMENT.—No special treatment specifically directed at the diseased condition has proved to be of any avail. Both drugs and organotherapy have proved equally useless. All that can be done in these cases is to place the patient under the general conditions of hygiene and environment which conduce to the most rugged normal development.

PROBLEMS AND RESEARCH.—The problem of the etiology and pathogenesis of this disease is so obscure that little progress has been made. Recent researches have been carried out upon the metabolism in patients with osteogenesis imperfecta, but these are of scientific interest rather than of any practical value.

CHONDRODYSTROPHY

(Achondroplasia)

This is a rather rare congenital disease characterized chiefly by an interference with the growth in length of the long bones. It has been recognized as an abnormality since the days of the Egyptians, and is of interest as being the cause of the most marked examples of dwarfism, most of the dwarfs which are exhibited in circuses and dime museums being specimens of this disease. The condition has often been improperly described as congenital or fetal rickets, but the lesions of the bones are entirely different.

ETIOLOGY—The abnormal growth of the bones begins during fetal life. The cause is entirely unknown, the instances in which an hereditary connection has been traced being so rare that this is probably not a factor.

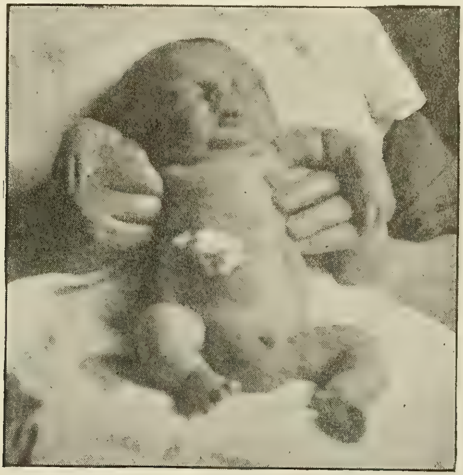
PATHOLOGICAL ANATOMY.—The essential lesion is more or less the reverse of that seen in osteogenesis imperfecta, in which the proliferation of cartilage is normal but ossification is diminished. In chondrodystrophy the process consists in interference with proliferation of the cartilage cells in the zones of growth at the ends of the diaphyses of the long bones. The lessened growth of cartilage through inhibition of proliferation of the cells is seen in all degrees. The result is interference with the growth in length of the long bones. On the other hand, the process of osteoblast formation, penetration, resorption and calcification, through which the cartilaginous tissue is converted into bony tissue, is entirely undisturbed. Furthermore, periosteal ossification is entirely normal and may even be excessive. The flat bones escape entirely.

The result of these disturbances is the formation of abnormally short and thick long bones, and premature synostosis. At times an abnormal connective tissue originating from the periosteum sinks into the epiphyseal zone of growth, the so-called periosteal lamella.

This causes an interference with growth in length more marked on one side of the bone than on the other, the result being curvature of the bony shaft. The width of the cartilaginous epiphyses varies, being in some cases lessened, in others increased, (the so-called hypo- and hyperplastic varieties).

The vertebrae are only slightly affected. The most marked interference with growth is seen in the humeri and the femora, but all the long bones of the extremities are affected to a variable degree. The marked prominence of the forehead and upper jaw seen in these cases, is due to the premature synostosis of the tribasalar bone (the two parts of the sphenoid and the sphenoidal process of the occipital bone). Normally this ossification does not take place until adult life, but in patients with chondrodystrophy it may begin during fetal life. This prevents a normal expansion of the base of the skull, and the prominence of the forehead is produced by the crowding upward and forward of the growth of the brain.

FIG. 251



Chondrodystrophia foetalis. Age 4 days

SYMPTOMS.—In the majority of cases of this affection the children are either still-born or die shortly after birth. Those who survive are delicate and have a low resistance during infancy, but if they reach childhood they may become strong and healthy.

The most striking feature of the clinical picture is the short arms and legs. Their length is greatly diminished in proportion to that of the trunk, and still more so in proportion to the circumference of the head and abdomen. The skin and soft parts hang on the limbs in deep folds, appearing too abundant for the shortened bones and suggesting sleeves or trousers which are much too long. The cranium is large, and may even measure above the normal average in circumference. The forehead is usually markedly prominent. The neck is plump and there is usually a good development everywhere of subcutaneous fat. There may be delayed closure of the fontanel, and moderately delayed dentition. The short fingers are nearly equal in length, and the angular separation of the fingers at the second joints gives the characteristic appearance of the "trident hand." In the early years there is muscular weakness and relaxation of the ligaments, so that the functions of walking and standing are delayed often until the third or fourth year. Later a marked lordosis develops, which with the prominent hips and abdomen, short extremities, and possible bowing of the legs, presents a very striking picture.

Mental development may be wholly normal but is usually somewhat below normal, although these dwarfs cannot be classed as defectives. They are usually easily amused and controlled, but do not often make markedly successful progress in their education. In adult life their sexual power is normal. They frequently live to a great age. The maximum height which they attain is often not more than 3 1-2 or 4 feet.

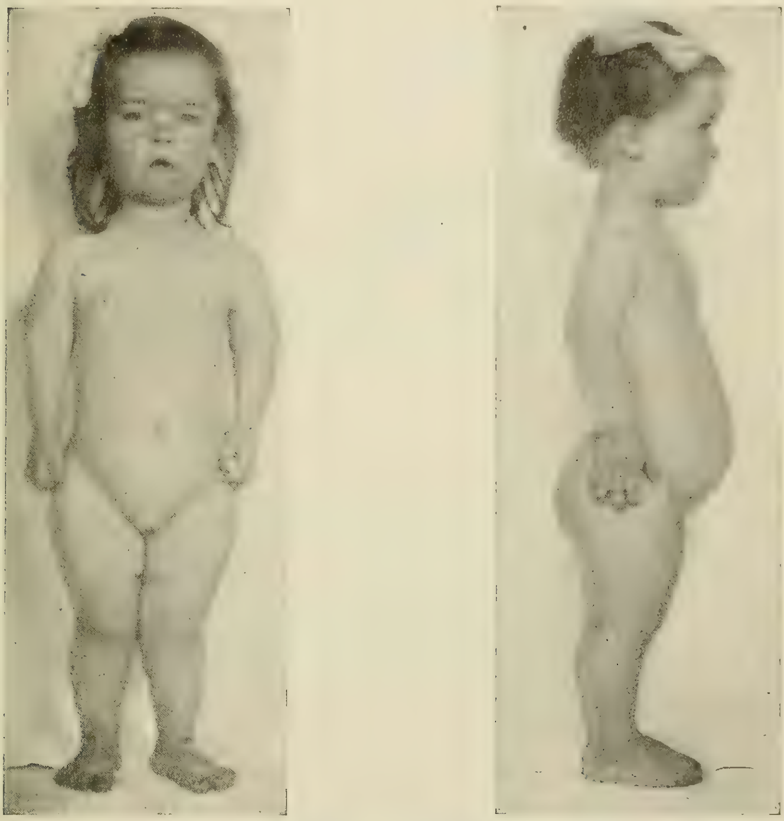
DIAGNOSIS.—The principal positive point in diagnosis is the marked shortening of the extremities. This with the characteristic appearances of the face, cranium and hands usually enables the diagnosis to be made without difficulty. The following conditions have to be considered in differential diagnosis.

Rickets.—Both rickets and chondrodystrophy have a prominent forehead, delayed closure of the fontanel, delayed dentition, broadening at the epiphyses, excessive sweating, normal mental development, and delay in walking and standing. The signs of rickets never develop so early in infancy as do those of chondrodystrophy. Furthermore, rickets does not show diminution of the length of the long bones, this being the most important differential point. The characteristic rosary of rickets is not seen in chondrodystrophy, and the enlargement in the region of the epiphysis in rickets is at the epiphyseal junction, while in chondrodystrophy it is the entire epiphysis which is enlarged.

In roentgenograms the appearances are entirely different. The epiphyseal lines instead of being broad and irregular as in rickets, are straight and much narrowed. The shafts of the bones are diminished in length and increased in breadth and density. The apparent overgrowth of the bony epiphysis may be so marked that its shadow appears to overlap that of the epiphyseal line.

Cretinism.—This condition is mistaken for chondrodystrophy because it also shows the principal diagnostic feature of the latter, namely, shortening of the length of the limbs. The shortening, however, is not so marked in cretinism as in chondrodystrophy. Furthermore, all the other features characteristic of the cretin, such as marked retardation of mental development and the myxedematous thickening of the skin and mucous membranes, are absent. Certainty of differential diagnosis may be obtained from the characteristic features seen in roentgenograms of chondrodystrophy.

FIG. 253



Chondrodystrophia foetalis in a girl of 5½ years

Hydrocephalus.— Chondrodystrophy has been mistaken for hydrocephalus because it may show an increased circumference of the cranium with wide-open fontanels. The principal differentiating point is the shortening of the limbs in chondrodystrophy, which is entirely absent in hydrocephalus.

Osteogenesis Imperfecta.—In some cases of this disease the femora may be shorter than normal, but it is easily recognized by the tendency toward multiple fractures, which are entirely absent in chondrodystrophy.

PROGNOSIS.—When the condition is first seen in early infancy, the chances for ultimate survival are rather bad on account of the delicacy and low resistance of these patients. In the few cases which are first seen after childhood has been reached, the prognosis as to life is good.

TREATMENT.—No known treatment has any influence upon the condition. The use of thyroid extract, or any other form of organotherapy has proved to be entirely without effect. General hygienic treatment is all that can be done.

ACUTE OSTEOMYELITIS

Acute infectious osteomyelitis is an acute infection involving the bones and joints. It has been described under various other names, such as *acute arthritis of infants*, *acute purulent synovitis of infants*, *acute epiphysitis*, and *pyemia of the bone*.

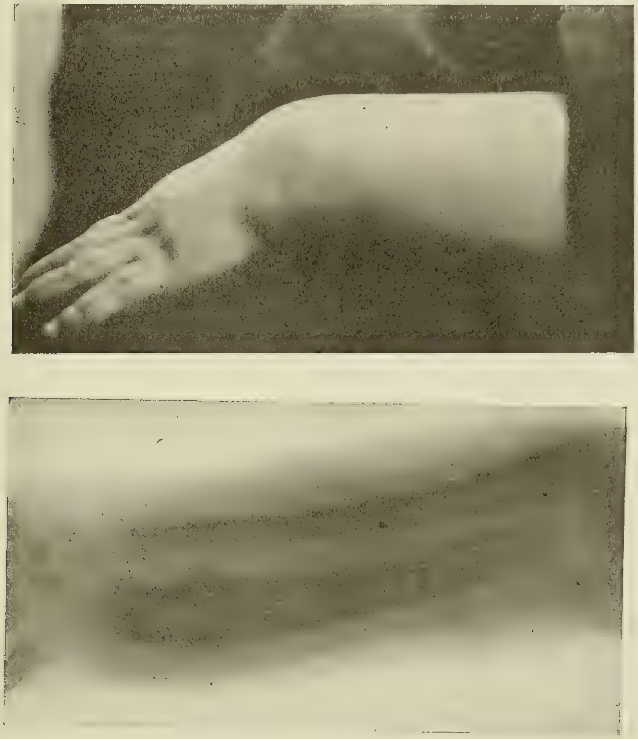
ETIOLOGY.—Nichols has shown that acute infectious osteomyelitis is an acute inflammation of the bone due to any one of a number of pathogenic organisms, hence osteomyelitis is not a specific disease, but belongs to the group of septic pyemias. The staphylococcus pyogenes aureus is the organism that most commonly produces the disease. An early period of life, and the acute infections predispose to osteomyelitis. Traumatism must be recognized as an occasional exciting cause.

PATHOLOGICAL ANATOMY.—In infants the disease commonly originates from a septic infection of the umbilicus or pharynx. The process attacks the bone-marrow primarily, and the joints become secondarily involved if the process originates in or extends to the end of the bone and is not limited to the shaft. The joint infection may come either from a separation of the epiphysis, which is wholly or in part intracapsular, or from a growth of pyogenic organisms along the blood- or lymph-channels of the epiphysis, frequently setting up an osteomyelitis of the ossified portion of the epiphysis and spreading thence into the joint. The two affections, osteomyelitis and arthritis, therefore, represent different phases of bone infection, rather than different diseases of the bone. The morbid process sometimes involves the entire structure of the bone, including the periosteum.

Osteomyelitis begins as a hyperemia of the bone-marrow. Later, suppurative foci of a dull-yellow or grayish color appear, while in severe cases the entire marrow becomes purulent and the Haversian canals of the cortical portion become filled with pus. Metastatic abscesses and thrombosis of the veins of the marrow may follow. When the infection enters the joint, the cartilage is softened and destroyed and an abscess is formed. Osteomyelitis may, however, be the cause not only of suppurative but of a simple inflammation of the joint.

SYMPTOMS.—The onset is sudden and sometimes marked by a chill. There are severe constitutional symptoms, fever, and intense pain, usually sharply localized. The part affected is swollen, reddened, and tender, and if a joint itself is involved, the swelling which is at first tense soon becomes fluctuating. From the beginning of the disease the signs of sepsis are manifest, with high intermittent fever, rapid pulse, pronounced leukocytosis, and great prostration. In exceptional cases these severe constitutional symptoms may not be present. In rapid cases death from general sepsis may occur in three or four days, but the disease is generally prolonged for one or two weeks.

FIG. 254

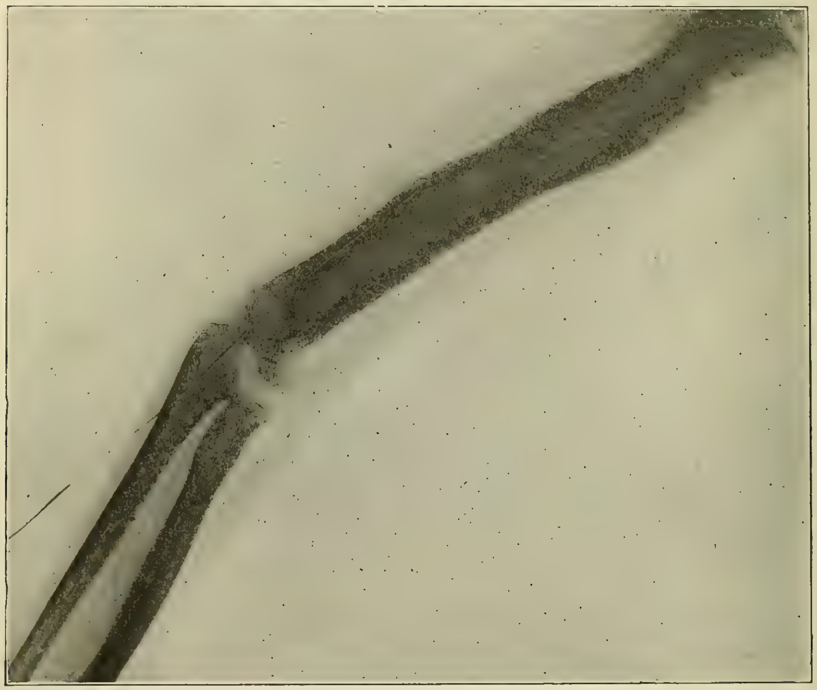


Case of osteomyelitis of the radius, the radiograph showing a marked periosteal thickening, as well as a fairly well defined focus of rarefication in the medulla of the bone itself. The extreme proximal end of the bone, as well as the distal epiphysis, is apparently not affected.

DIAGNOSIS.—The differential diagnosis is to be made chiefly from the articular form of rheumatic fever, from which it is to be distinguished by the surrounding soft parts being much less involved and the general symptoms much more severe in osteomyelitis, and, if the disease occurs in the first two years, by the rarity of rheumatism at so early a period.

From tuberculosis, osteomyelitis is to be differentiated by the slow onset of the former, and by the fact that the primary lesion generally shows itself in the shaft of the bone in the latter, while in the former the epiphysis is usually first attacked. The process of osteomyelitis, may, however, attack the epiphysis and extend to the joint. The presence of a leukocytosis in osteomyelitis is also of great aid in the diagnosis.

FIG. 255



Osteomyelitis of the humerus

PROGNOSIS.—The prognosis is very unfavorable unless immediate and radical surgical interference is carried out.

TREATMENT.—The treatment is essentially surgical. When osteomyelitis of the shaft of a long bone is found at operation, all of the affected portions should be removed, even if a considerable portion of the shaft is taken, experience having shown that new bone will soon form, and that a serviceable limb is the usual result in the leg and forearm.

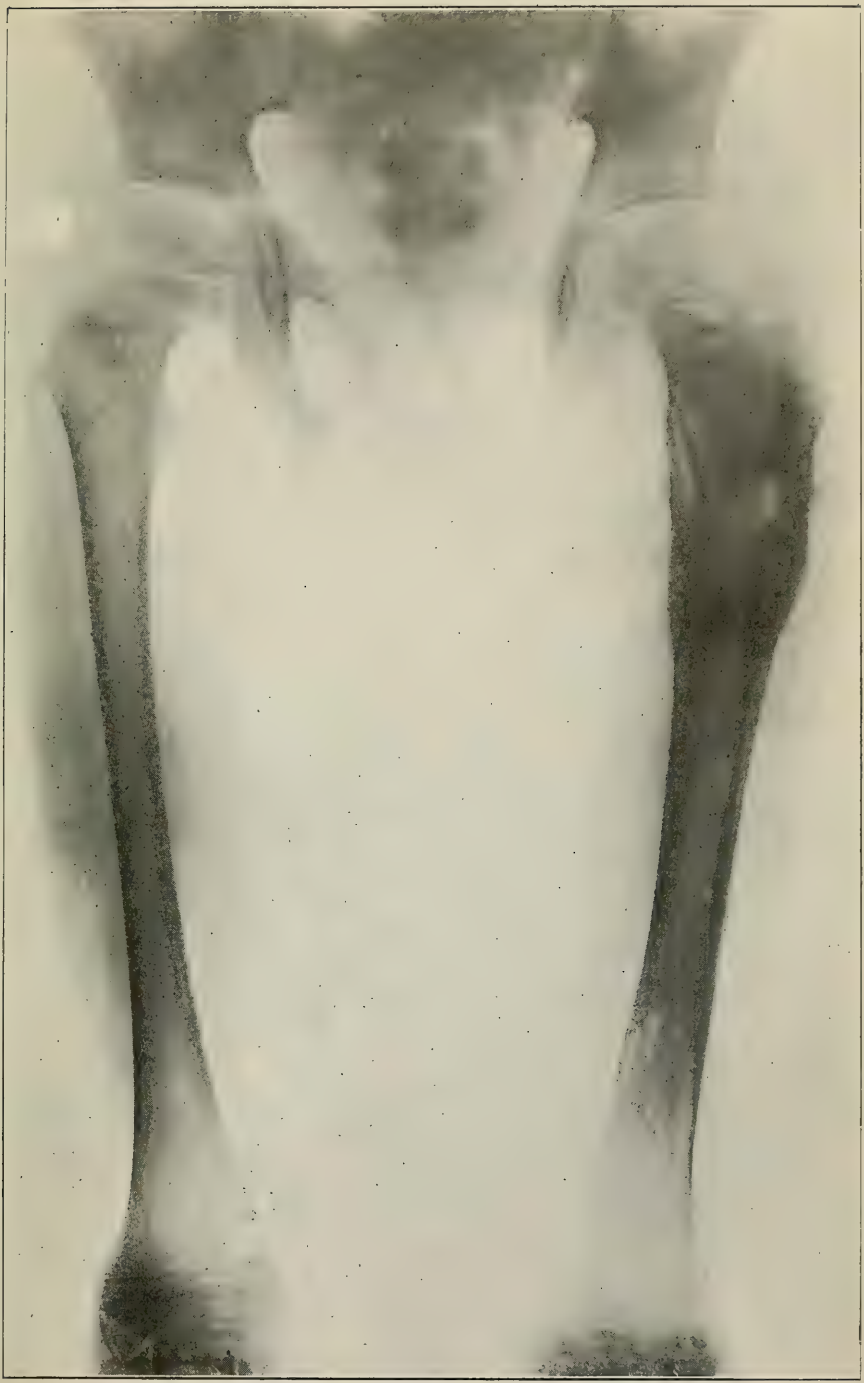


FIG. 255—Osteomyelitis of the femur

Prevention of septic infection of the umbilical cord, and of the wound after the cord has fallen off, is important as a matter of prophylaxis.

ACUTE PERIOSTITIS

This is an inflammation of the periosteum.

ETIOLOGY.—There are two forms, traumatic and infectious. In the traumatic form the inflammation is due to a mechanical injury and is seen most often on the shin bone. In the infectious form the etiology is the same as in acute osteomyelitis, the only difference being that the process begins in the periosteum rather than in the medullary cavity of the bone. The process is usually secondary, and appears as a complication of other recognized acute infections. In cases which are apparently primary, there is probably an unrecognized focus of infection elsewhere, although it is possible that under certain circumstances, possibly with trauma as a preceding cause, organisms circulating in the blood may locate in the periosteum as a point of least resistance.

PATHOLOGICAL ANATOMY.—The purely traumatic form is not suppurative. The lesion consists in hyperemia and plastic exudation. In the infectious form the process is a subperiosteal suppuration.

SYMPTOMS.—In the traumatic form the only symptom is marked tenderness over the bone, most commonly over the shin. There is also a moderate amount of swelling. The condition is very similar clinically to the periostitis of syphilis, the principal differentiating point being the history of the tenderness having followed trauma. In traumatic periostitis the tenderness and swelling may persist as long as several weeks.

In the infectious form the symptoms closely resemble those of acute osteomyelitis. There is fever and the usual constitutional disturbance which accompanies acute infection. The symptoms, however, are usually much less severe than in acute osteomyelitis. On examination, an area of acutely tender swelling is found over some portion of the bone. Later, fluctuation is detected.

PROGNOSIS.—Acute periostitis is a less severe and dangerous disease than is acute osteomyelitis, and does not often lead to general sepsis. With proper treatment rapid recovery should take place.

TREATMENT.—In the traumatic form the application of cold in the form of an ice-bag or cold compresses is indicated. The area should be most carefully protected from a mechanical injury.

In the infectious form, the treatment is essentially surgical, consisting in incision and drainage.

INFECTIOUS ARTHRITIS

(Still's Disease)

ETIOLOGY AND PATHOLOGICAL ANATOMY.—In the great majority of cases in which the joints are affected in children, the pathological process is due to infection. This infection is due to the presence in the body of one of the specific organisms, or a group of organisms, and the affection of the joints in most cases occurs in the course of the general specific disease. The primary focus of infection may be in any part of the body, as the tonsil or the ear. The arthritis may be due to the presence of the specific organism in the joints or to the action on the joints of its toxins alone. In the former case the local process is more acute and severe. It is thus seen that under the general term infectious arthritis should be included a very large number of diseases.

The character of the joint lesions depends upon the special organism present, and while in certain instances, such as infections from the tubercle bacillus and in syphilis, the anatomical conditions can often be recognized, yet in most cases the diagnosis is made by the occurrence of the arthritis in the course of one of the specific infections, such as rheumatic fever gonorrhea, scarlet fever, or typhoid fever. The diagnosis, symptomatology, and treatment of these cases of acute joint affection have been described under the especial diseases in which they occur.

Infectious arthritis may be the result of any of the pus producing organisms. The severity of the attack depends not only upon the special organism, but upon its virulence, whether it is actually present in the joint, and upon the idiosyncrasy of the individual child. Such organisms as the pneumococcus, streptococcus, staphylococcus, influenza bacillus, bacillus of dysentery, and many others, may produce an arthritis. We have not as yet, in quite a large number of cases, determined the especial pathology and symptoms belonging to the several specific infecting organisms.

SYMPTOMS.—There are cases in which the result of infection is a chronic lesion of the joints. It is this group of cases in which Still has done such excellent pioneer work, and his description of the symptom complex representing them is of much value. We should, however, recognize that etiologically there is no such disease as "Still's disease," but that a number of infections may produce the group of symptoms which has been called by his name, which should properly be referred to as *chronic infectious arthritis*. Looking at the subject from this point of view it is easily understood that all of the symptoms described by Still need not be especially marked in every case, and that different symptoms of infection may arise in certain cases according to the nature or virulence of the infection. Consequently no fixed symptomatology will cover all cases. The spleen is more apt to be enlarged in children than in adults owing probably to its greater reaction to toxemia in early life. The symptoms are those of infection and may begin acutely with fever and chills or insidiously with stiffness and gradual enlargement of the joints, which assume an elastic fusiform shape without bony change.

Still's description of the various phases of this group of infections in children covers the ground as well as can be done until we have a more precise knowledge of the characteristic manifestations of the individual infecting organisms.

It is as follows:—

The joints earliest affected are those of the hands, wrists, and cervical spine, the ankles, elbows, fingers and other joints being subsequently involved. The affection of the joints is symmetrical. The enlargement looks and feels more like a general thickening of the tissues around the bones than a bony enlargement, and is correspondingly smooth and fusiform without bony irregularity. There is no osteoblastic growth even after years. There is never bony grating. There is no tenderness or redness except in very acute cases. Pain is absent as a rule, except on motion, when it may be present. There is always limitation of motion. There is no tendency to suppuration or bony ankylosis. The muscles which move the diseased joints show early and marked wasting without change in the electrical reactions. The most distinctive feature is the enlargement of the lymphnodes, which is general, but affects primarily and chiefly those related to the affected joints. The glands are discrete, hard, not tender, and show no tendency to break down. Their size varies with the acuteness of the symptoms in the joints. Enlargement of the spleen is almost always present, the amount of the enlargement varying directly with that of the glands and the acuteness of the joint symptoms. There is generally a moderate diminution in the number of red corpuscles, often associated with a disproportionate diminution in the amount of hemoglobin. The febrile attacks are not usually associated with any clinically demonstrable exacerbation of the joint trouble. The urine shows nothing important. Slight exophthalmos is sometimes present. The heart shows no evidence of valvular disease, although adherent pericardium has been found several times at autopsy. The course is slow. Improvement may take place for a time under treatment or spontaneously. The disease soon progresses again, however, until a condition of general joint disease is reached which seems to be permanently stationary. It is not fatal, the few recorded deaths having been due to complications. Pathologically, the joints show marked thickening of the capsule and of the connective tissues just outside it. There is also thickening and vascularization of the synovial membrane. Fibrous adhesions are sometimes present. The cartilages may show pitting at their margins from the pressure of little processes of the thickened synovial membrane, but are otherwise healthy.

The X-ray is of great importance in the differential diagnosis. The important feature is the entire absence of changes in the bones.

TREATMENT.—Serum therapy has not yet been sufficiently successful to be recommended at present. The treatment of each case must be symptomatic and directed in the beginning to the acute symptoms. It is well to bear in mind that the salicylates are not indicated and may do harm by their depressing effects. The later treatment is to build up the general health with fresh air, good food, and tonics. Many cases can be helped by an expert orthopedist.

CHRONIC ATROPHIC AND HYPERTROPHIC ARTHRITIS

Although a large amount of work has been and is being done in connection with diseases of the joints in children, the whole subject is still in the process of reconstruction so far as etiology and classification are concerned. As in other diseases, such as of the mouth and of the gastro-enteric tract, various names have so long been used to represent the same disease, and various diseases have so often appeared under the same name, that much confusion has necessarily resulted, and the work of excellent investigators has thus been often invalidated.

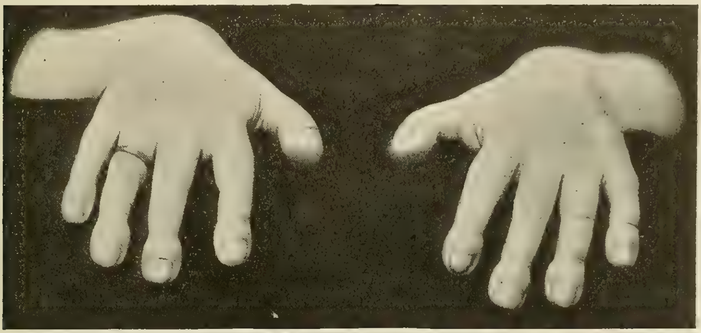
It would be better to discard all the unmeaning names were it not that they are so generally used that they must still be recognized as having a place in nomenclature. At the same time we can explain wherein they are lacking and substitute for them such provisional names as express our present knowledge of the subject. In this sense we should look upon as misleading, in connection with joint affections in children, such headings as rheumatoid disease, Still's disease, arthritis deformans, arthritis of infants, and a number of others.

The question which has arisen, and which is still not absolutely settled, is, whether a certain class of cases, in which the symptoms are those of a slowly developing, progressive disease affecting the joints or their immediate neighborhood, represents a number of types of the same disease sometimes called by such names as rheumatoid arthritis and arthritis deformans, or whether this class represents separate diseases. Our knowledge of the etiology of these cases is practically *nil*. The postmortem findings in all these chronic types have necessarily been misleading and unsatisfactory, as in most cases it is only the terminal pathologic process which can be studied, and these terminal processes might well simulate each other, although differing markedly in their early lesions.

A careful study of the symptoms and physical condition of the parts affected, combined with the evidence of X-rays from the beginning and following the progress of the especial case, has given the most valuable information regarding these conditions and leads us to believe that we are dealing with different processes rather than with different phases of the same process.

Although this class of cases is at times met with in children, it is extremely rare and should be studied from works devoted to adults and to orthopedics rather than to pediatrics; it will, therefore, be dealt with briefly and with the intention of showing how the treatment differs in the two chief types. In this connection it is well to state, merely because the necessity of a differential diagnosis may possibly arise, that *chronic gout* may occur in childhood; also that what has been called so universally *villous arthritis* is in children a secondary condition and need not be considered. The remaining types of slow, progressive deformities of the joints have been extensively studied in England and in America with the result that practically they are narrowed down to the two chief types referred to above as *atrophic* and *hypertrophic*. The evidence is in favor of their being two diseases rather than two types of the same disease. Whatever names may be used in the future, the condition in one group of cases is essentially one of *atrophy* and in the other of *hypertrophy*.

FIG. 256



Chronic pulmonary osteo-arthropathy in a boy 3¾ years of age. Total chronic consolidation of left lung from influenza of several months' duration. Radiographs of the fingers and toes showed the terminal phalanges as well as the soft parts to be both elongated and thickened.

The atrophic cases have been designated by such names as *rheumatoid arthritis* and *arthritis deformans*. The cause of the disease is unknown; it has been suggested that it is of a trophic nature dependent upon some obscure disturbance of metabolism. All the joints may be affected slowly and progressively, but the fingers are usually affected early. There is swelling of the soft tissues about the joints, with subsequent atrophy of the bony and cartilaginous parts of the joint. The diagnosis is made chiefly by means of the X-ray. The treatment is essentially to increase the metabolism by massage, fresh air, good food, and tonics. When this can be done the disease is often arrested and its severity mitigated, but it usually goes on for years.

The hypertrophic cases are those which have usually been designated by such names as *osteo-arthritis*, *chronic rheumatic arthritis*, and *arthritis deformans*. The cause is unknown. It may be a local or general process characterized by swelling of the joints, with hypertrophic changes in the bones and cartilages of the joint.

Heberden's nodes, to be distinguished from the freely moving urate of soda deposits of gout, may occur in the finger-joints.

Atrophy of the surrounding soft tissue with an end result of ankylosis of the joint may occur. The diagnosis is made chiefly by the X-ray. The treatment is fixation in the active stage and, later, orthopedic measures.

TUBERCULOSIS OF THE BONES AND JOINTS

Chronic tuberculous disease of the bones and joints belongs especially to the domain of orthopedics, and is exhaustively described in all orthopedic textbooks. A thorough description of bone-joint tuberculosis would occupy much space which ought to be devoted to more exclusively medical diseases. For this reason I shall make no attempt to give a complete description of these diseases, but shall confine myself only to outlining their general diagnostic features.

ETIOLOGY.—The pathogenesis of tuberculous disease of the bones and joints, and the relation of the lesions to tuberculous infection in early life, have been described in the general article on tuberculosis in Volume II. This manifestation of tuberculosis is essentially a disease of childhood rather than of infancy. It occurs most often between the ages of three and eight years.

In 1,344 cases of hip disease, 1,000 occurred under fifteen years of age. (Bradford and Lovett.) A series of cases from the New York Dispensary showed that 115 occurred under three, 316 from three to five, 509 from five to ten, 140 from ten to fifteen, and 98 over fifteen years. This may be taken as fairly representative of the general liability to the disease at different ages. At the Children's Hospital, Boston, from 1869 to 1893, 3,820 cases of tuberculosis of the joints were as follows.

TABLE 73

Spine	1964
Hip	1402
Ankle	300
Knee	104
Wrist	20
Shoulder	15
Elbow	15

These figures are similar to those reported by other American authors.

Tuberculosis of the bones and joints represents a stage of the relations between host and parasite characterized on the part of the host by comparatively high powers of resistance, and on the part of the parasite by efforts to escape from the body of the host before this high resistance shall have proved fatal to the continued life of the parasite. Hamburger and other foreign writers classify bone-joint tuberculosis with phthisis as representing a " tertiary " lesion of tuberculous infection in early life, as opposed to the primary lesions at the point of invasion and in the adjacent lymphnodes, and such secondary lesions as bronchopneumonia, meningitis, pleurisy, peritonitis, and general miliary tuberculosis. Under this view it bears the same relation to the battle between host and parasite as do the tertiary lesions of syphilis.

The localization of the tuberculous process in the bone can be explained on the basis of a relatively less local tissue resistance. Traumatism, however, has been generally accepted as an important etiological factor. The bones of active children are constantly subjected to various mechanical shocks, and their delicate structure affords an opportunity for the formation of a local mechanical lesion which might easily become a nidus for the tuberculous infection. Other conditions, such as acute infectious diseases, are also known to play a contributing rôle in all extensions of tuberculous infection beyond the limits of the primary lesion.

PATHOLOGICAL ANATOMY.—Tuberculosis of the joints in children, as well as in adults, is now much more uniformly regarded as being of bony origin than was formerly supposed. The existence of a primary synovial tuberculosis in children must be regarded as extremely rare, and only to be demonstrated by the examination of all parts of the bones entering into the formation of a joint. The finding of a tuberculosis of the synovial membrane at operation, without an obvious bony lesion, cannot be accepted as proof of primary synovial disease.

The original statement of Volkmann is, " The fungus inflammation of the joints in children and adults begins generally not at all as an arthropathy, but as a pure osteopathy, with a very circumscribed condition of tuberculous osteitis." Nichols, in the examination of 120 tuberculous joints from children and adults, did not find one joint where, if all the bones entering into the joints were sawed open in thin layers, one or more old bone foci were not found.

The process by which joints become affected by tuberculous disease is as follows A tuberculous focus develops in the spongy tissue of the bone, usually in the epiphysis, and by an extension of this process the bone is destroyed in the line of least resistance, which is usually towards the surface of the joint. When perforation of the joint by this focus occurs, the tubercle bacilli obtain access to the joint cavity and a tuberculous synovitis begins. From this point destruction in any direction is, of course, possible. Under favorable conditions of rest and separation of the surfaces of the joint by traction, in certain cases, Nature tends to limit the process by repairing and replacing the tuberculous tissue by the formation of fibrous tissue which grows into and replaces the tubercular material. The soft parts around the joint are, of course, secondarily affected, and abscesses are a frequent complication.

The contents of a tuberculous abscess may be sterile so far as pyogenic organisms are concerned, or they may contain, especially in their later stages, pyogenic organisms. Tubercle bacilli are to be found in these abscesses in only about one-third of the cases, and, as a rule, only after prolonged search. Inoculation experiments must be relied upon in the majority of cases to establish their presence.

HIP JOINT DISEASE

SYMPTOMS.—The onset is usually very gradual and insidious, and the first symptoms are often so slight that they do not arouse suspicion in the minds of the child's parents that anything serious is wrong. The first symptom noted is usually a slight lameness due to stiffness of the joint. This is apt to be most marked in the morning, and in the early stages often wears off entirely during the day. In some cases children show instead of a lameness, a disinclination to walk. There may be some tenderness about the hip even in this early stage of the disease. The next symptom is a complaint of pain by the patient, who refers it almost never to the hip, but rather to the knee or to the outer surface of the thigh. Later on the pain may take the form of the sudden sharp, " starting " pains at night, which may cause the child to cry out sharply without waking, or to wake up with a cry. Finally, lameness becomes constant and more severe. The duration of these early symptoms before more serious signs of the disease develop is very variable, being sometimes only a few weeks, but more often four or five months, and sometimes the symptoms come and go in a most perplexing way for a much longer time.

PHYSICAL SIGNS.—Any child with any of the symptoms mentioned above should be stripped and subjected to a very thorough examination. In the first place, the contour in the region of the hip should be noticed. Significant signs of hip disease are a prominence of the trochanter and a broadening of the gluteal region, with shortening of gluteal fold. Often a single instead of a double gluteal fold is noted on the affected side. In some cases there may be a certain amount of palpable thickening about the trochanter, or some tenderness to pressure.

The next point to be noted is the attitude and gait of the child. The lameness is apt to be of such a character that the child in walking obviously favors the affected side.

The next point in suspected cases is the examination of the function of the hip joint. Limitation of motion in some direction is almost invariably present after the disease has existed for a few weeks, and is the most important of all the diagnostic signs. The child should be placed flat upon its back on a table, and passive movements of the hips should be made in all directions, with a careful comparison of the range of movement of the two sides. Movement on the affected side is limited by muscular spasm; the limitation may be in any direction, but is most often in abduction, rotation, or extension. Careful note should be taken as to whether there is any muscular atrophy, and finally measurements should be taken for shortening, although this sign is not present usually until the later stages of the disease.

THE LATER STAGES.—As the disease progresses, after a period of weeks or months a permanent deformity takes place, due to muscular spasm. The leg assumes an abnormal position, the most typical in this stage being slight flexion of the thigh on the body, outward rotation of the thigh, and eversion of the foot. In this stage of the disease, there may be inability to walk, or lameness may be very marked. Acute exacerbations are liable to occur from time to time characterized by a marked increase of pain, excessive tenderness and pain on motion, and complete inability to walk. Abscesses may form at any point about the hip-joint, and these finally open externally with a discharge of pus; they are followed by the usual unsightly cicatrization characteristic of tuberculous suppuration.

In a still later stage of the disease, the deformity becomes still more marked and the limb becomes fixed in its abnormal position. The typical position in the most extreme cases of hip disease is that of marked flexion and adduction, with inward rotation of the thigh. In this stage the trochanter is usually found on the upper surface of the ilium above Nelaton's line. There is marked shortening of the leg, no motion at the hip, the process terminating in ankylosis. Marked lordosis of the spine is also usually present in this stage of the disease.

DIAGNOSIS.—The importance of making an early diagnosis in cases of hip disease cannot be overestimated. The physician should remember that the appearance in a child of any lameness, no matter how slight, is a very suspicious sign, and should lead to the most careful examination. When, in addition to lameness, there is any pain referred to the knee or thigh, or any starting pain at night, the case is still more suspicious. The most important diagnostic sign in this stage of the disease is any limitation by muscular spasm of the movements at the hip-joint. A very important point in differential diagnosis is the very gradual evolution of the symptoms and their apparently trivial character at first. The tuberculin reaction is usually positive in all forms of tuberculosis of the bones and joints, and its presence is valuable confirmatory evidence. Still more valuable at times in the recognition of the tuberculous nature of the process, is the evidence obtained by X-ray examinations, which often even in the earliest stages of the disease disclose the presence of a tuberculous focus in the bone near the hip.

In differential diagnosis the conditions to be considered are, a strain of the joint, muscular rheumatism, chronic non-tuberculous arthritis, poliomyelitis, periostitis of the shaft of the femur, phlegmonous inflammation about the hip, and tuberculosis of the lumbar spine The diagnosis from these conditions is based on the finding of the peculiar features characteristic of hip disease enumerated above. In the later stages of the disease the diagnosis is usually obvious.

PROGNOSIS.—The process may be arrested at any stage. The prognosis must be considered both as to life and as to function of the joint. While tuberculosis of the bones and joints is not one of the most fatal forms of tuberculosis encountered in early life, yet it must be remembered that the prognosis of tuberculous infection in general in children is not very good, on account of the liability to widespread fatal lesions, especially tuberculous meningitis and general miliary tuberculosis. The tendency toward the development of these serious manifestations of the disease is much less in childhood than in infancy, but nevertheless is still much greater than in adult life. Furthermore, the exhaustion and lessened resistance from the prolonged suppuration of bone-joint tuberculosis, and the occasional occurrence of amyloid degeneration of the viscera are additional factors affecting the prognosis not immediately connected with the tuberculous infection. The hospital mortality of patients with hip disease is about 25 per cent. The outlook as to the function of the hip-joint depends very largely upon the stage of the disease in which treatment is begun, and upon the skill with which the treatment is employed. With early recognition of the nature of the disease and the institution of proper treatment, recovery often occurs without lameness or any other impairment of the joint functions. In less favorable cases there is usually a variable degree of lameness, which may be due only to shortening and muscular atrophy, but which may also be due to some permanent limitation of the movement of the joint. In unfavorable cases, or cases coming under treatment in an advanced stage of the disease, there is always marked shortening, deformity and lameness

TREATMENT.—The treatment of hip-joint disease is essentially surgical. Every patient should at once be placed in the hands of a skilled orthopedic surgeon. The general measures for the treatment of tuberculosis are, of course, indicated.

TUBERCULOSIS OF THE SPINE

(Caries of the Spine; Pott's Disease)

SYMPTOMS.—The principal symptoms of tuberculosis of the spine are. (1) referred pain; (2) rigidity of the spine; (3) peculiarities of gait and posture; (4) deformities of the spine; (5) the formation of " cold " abscesses; (6) paralyses.

The referred pain is due to irritation of or pressure upon the posterior nerve roots. The pain is referred to various parts of the body, following the distribution of the spinal sensory nerves. It is one of the earlier symptoms. Rigidity of the spine is due to muscular spasm, which in turn is produced by an effort to protect the diseased area from jar and movement. The peculiarities of gait and posture seen in tuberculosis of the spine are produced in a similar way. The patient assumes postures tending to relieve the diseased area from pressure and to protect it from the effects of jar and movement. Involuntary muscular spasm and these peculiarities of gait and posture are also early symptoms of the disease. The deformities are produced by the destructive process in the bodies of the vertebrae, which causes a curved prominence or angular projection in some portion of the spinal column. This is a comparatively late symptom. Abscesses are produced when the tuberculous process goes on to softening and suppuration. They are apt to burrow in a direction determined by the lines of least mechanical and anatomical resistance, appearing in various localities according to the situation of the tuberculous process in the spine. Paralyses are due to pressure upon the spinal cord or motor nerve roots.

The onset of the disease is gradual and insidious. The first symptoms usually noted are, referred pain, rigidity of the spine, or peculiarities of gait or posture. Occasionally, however, these initial symptoms are slightly marked and the first symptoms noted are the appearance of an abscess or of evidences of paralysis.

CERVICAL DISEASE.—When the process is in the cervical spine, the pain is often referred to the front or sides of the neck; sometimes it is referred to the occipital region of the head, the complaint being of headache. The pain may be present and of a varying severity when the child is at rest, or may only be complained of on active or passive movement of the head. At times, especially in acute exacerbations, the pain produced by movement is very severe. Muscular spasm in the early stages of the disease most often manifests itself as a slight torticollis. There may be a fixation of the head by a tonic spasm of all the muscles of the neck. In some cases the child actually steadies its head with its hand in order to prevent movement. All these symptoms develop very gradually and are persistent. Occasionally, however, they are overlooked, and the first thing which attracts the attention is the respiratory obstruction of a retropharyngeal abscess, or the appearance of progressive weakness of the lower extremities, which is the first sign of a developing paraplegia. Deformity is a late sign in cervical disease, often not appearing for many months, if at all. Kyphosis is only seen when the process is situated in the lower cervical region. The most common deformity is a settling of the head between the shoulders, with a broadening and thickening of the neck. Sometimes there is an anterior prominence in the upper cervical region which can be detected by digital exploration of the pharynx.

DORSAL DISEASE.—When the tuberculous process is in the dorsal spine, the pain is either referred to the abdomen or takes the form of intercostal neuralgia. The children walk very carefully, obviously favoring the spine which they hold erect and stiff. There is often at first a slight lordosis rather than a kyphosis. In the very early stages of the disease, children are apt to sleep face downward, and during the daytime they often like to lie face downwards across the chair or the nurse's lap. Deformity is much more constant and typical in dorsal than in cervical caries, and appears much earlier in the course of the disease. It takes the form of a kyphosis. At first the prominence may be a curvature, but later there is apt to be a typical angular deformity with especial prominence of one vertebral spine. There is a compensatory lordosis in the lumbar region. Abscess and paralysis when present, are usually comparatively late symptoms.

LUMBAR DISEASE.—When the tuberculous process is in the lumbar spine, the pain is most frequently referred to some part of the lower extremities, such as the hip, or outer aspect of the thigh or knee; at times it is referred to the groins or buttocks. Lameness is also present at times, giving rise to a suspicion of hip disease. There is often a tilting of the pelvis to one side. The child's gait and attitude are characteristic. He shows a disinclination to stoop down, and gets up awkwardly from the floor, obviously favoring the spine. He walks stiffly with short steps, holding the spine carefully erect with the shoulders back. Deformity is neither so early nor so marked as in disease of the dorsal spine. Curved kyphosis is apt to precede typical angular deformity. Psoas abscess may precede the appearance of deformity. Its premonitory symptoms are lameness and flexion of one thigh, and its existence is recognized by the presence of a palpable fluctuating tumor in the deep iliac fossa, or at the inner aspect of the upper part of the thigh.

PHYSICAL EXAMINATION.—The physician should have the child stripped and should note carefully all peculiarities of gait, posture and attitude. He should cause the child to pick up objects from the floor, and to lie down on the floor and get up again. This will disclose whether the spine is held rigidly and favored, and whether voluntary movements cause pain and discomfort or are accompanied by muscular spasm. It will often be noted that the child shows a disinclination to pick up objects from the floor, or that he gets up awkwardly. Characteristics of the gait and attitude described above will easily be clearly apparent if present.

The flexibility of the cervical spine is best tested with the child lying on its back on a table. Passive movements of the head can then be made, and it can be noted whether they are accompanied by muscular spasm or by pain. Flexibility of the dorsal and lumbar spines are best examined by turning the child over on its face. The body can then be lifted by the heels and the flexibility of the spine noted. Care must be taken, however, not to produce pain in this examination. Any muscular spasm or rigidity of any portion of the spine will be clearly apparent.

The physician should then look for deformity. In the early stages the kyphosis may be only a general curving, but later the more typical knuckle is present. This is caused by a projection of a single vertebral spine, and is usually most marked when the disease is in the upper dorsal region. Any slight projection which does not disappear when the child is suspended is suspicious. Tenderness to pressure along the spine is rarely present and is not an important symptom.

The physician should then examine for evidences of abscess formation. This should include careful inspection along the course of the spine, deep palpation in the iliac fossae, examination of the inner surface of the thigh, and digital exploration of the pharynx. The extremities should be carefully examined for evidence of paralysis.

Abscesses are seen in about 20 per cent of the cases of tuberculosis of the vertebrae. They are the typical cold abscesses. With disease of the cervical spine, the abscess may be retropharyngeal, retroesophageal, or may open above the clavicle. In disease of the dorsal spine, the abscesses are most apt to appear in the dorsal region, or above the iliac crest behind. In disease of the lumbar spine, the most common form is the typical psoas abscess, which opens either in the groin above Poupart's ligament, or at the upper inner aspect of the thigh. Paralysis is seen most often in disease of the cervical or upper dorsal spine, occurring in about half of these cases. It is rare when the tuberculous process is situated below the middle dorsal region.

COURSE OF THE DISEASE.—Tuberculosis of the spine is very chronic and runs a course lasting for months or years. Exacerbations and remissions are frequently seen. Exacerbations and relapses may be caused by trauma, by improper treatment, or by the early discontinuance of treatment. During an exacerbation, pain on motion is markedly increased, and an abscess or paralysis may be produced.

After a period varying from one to four years repair begins. The result of the process of repair is a more or less marked ankylosis and permanent deformity.

DIAGNOSIS.—The diagnosis of tuberculosis of the spine in its early stages depends upon the finding of the characteristic symptoms of referred pain and spinal rigidity mentioned above. The insidious onset and chronic nature of the disease is the most important diagnostic feature. The tuberculin reaction is usually positive in this form of tuberculosis. Roentgenograms are of great aid in the recognition of the nature of the process in the bones at any early stage.

After deformity has developed, Pott's disease must be differentiated from rachitic kyphosis and from rotary lateral curvature of the spine. The kyphosis of rickets is usually seen in children under eighteen months of age, a period of life at which Pott's disease is very rare. The angular prominence or knuckle characteristic of caries of the spine is not seen in rickets. Further confirmation of the diagnosis of rachitic kyphosis may be obtained from the finding of other characteristic signs of rickets, such as the rosary and the enlargement of the radial epiphyses. The most important differentiating point, however, is the absence of any spinal rigidity in rickets, the spine usually showing rather an abnormal mobility. This also applies to the various conditions in young infants in which a curvature of the spine is produced by malnutrition and muscular weakness.

Rotary lateral curvature is not seen in young children except in connection with rickets. It is seen most often at about the age of puberty. The early stages of some cases of tuberculosis of the spine may present a deformity resembling that of incipient rotary lateral curvature. The latter deformity, however, is entirely unaccompanied by pain or by any limitation of the mobility of the spine.

PROGNOSIS.—The prognosis as to life in tuberculosis of the spine cannot be definitely stated. It is that of tuberculosis in general at the age of the particular patient. The chances in favor of survival are probably about five to one, the causes of death being the tendency toward widespread secondary tuberculosis, the exhaustion from prolonged suppuration, amyloid degeneration, and so forth. Sudden death has been known to occur from pressure upon the cord in the upper cervical region, or from the pressure effects of retropharyngeal abscesses or abscesses in the posterior mediastinum.

The prognosis as to the amount of permanent deformity depends upon the situation of the tuberculous process, the stage at which treatment is begun, and the skill and thoroughness with which the treatment is carried out. The results are better when the disease is below the middle dorsal region than when it is situated high up. A great many patients recover with a very slight degree of deformity, and some without any whatever.

TREATMENT.—The general treatment of Pott's disease is that of tuberculosis in general, as described in Volume I. The local treatment should always be in the hands of an orthopedic surgeon.

TUBERCULOSIS OF THE KNEE JOINT

(Knee Joint Disease; Tumor Albus; White Swelling)

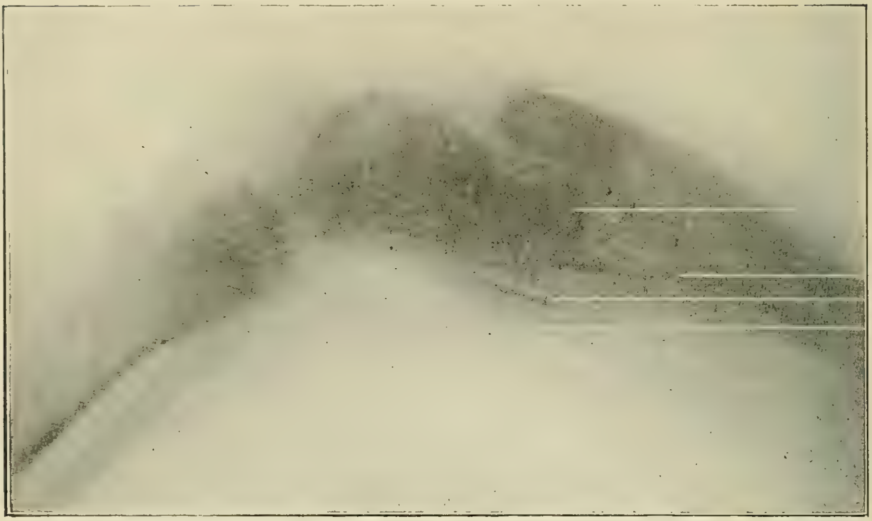
SYMPTOMS.—The principal symptoms of tuberculosis of the knee are, lameness, pain, and swelling. Like all forms of tuberculous joint disease, the process is very chronic and the onset is insidious. The first symptoms noted are usually slight lameness and slight stiffness of the joint. The child shows a tendency to walk with the knee slightly bent, and full extension is apt to produce pain. The symptoms are usually not constantly present in the earliest stages of the disease, but come and go. Finally the lameness and stiffness become constant, and pain is complained of, which is usually referred to the knee. Starting pains at night are much less common than in hip disease Examination in these early stages shows limitation of the motion of the joint.

Swelling is an early symptom in tuberculosis of the knee. At first the swelling involves chiefly the bone itself, one condyle, usually the inner one, being enlarged. Later the swelling involves the soft parts, causing a general fusiform enlargement which involves the entire joint and effaces the normal outlines. There may be some tenderness to pressure over the joint. There is often a noticeable atrophy of the muscles of the thigh and leg.

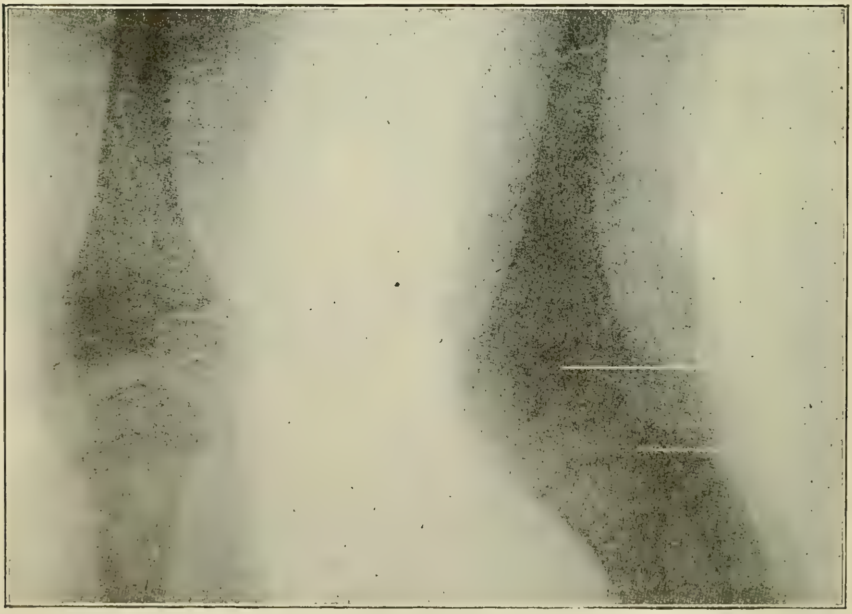
In the most advanced stages of the disease, deformity becomes marked. The joint may show signs of containing fluid. The knee is more or less flexed and somewhat rotated outward. As deformity increases the leg becomes more and more fixed in its abnormal position. Abscesses may form anywhere around the joint.

Tuberculosis of the knee resembles that of the hip both in duration and in the tendency to acute exacerbations and remissions. Exacerbations are caused by trauma or improper treatment, and are characterized by marked pain and tenderness.

FIG. 258



Osteomyelitis of the lower end of the femur, with spontaneous fracture. Boy 9 years old. A, lower end of upper fragment; B, upper end of lower fragment; C and D, portions of cortical bone which have been sequestrated, and were subsequently removed by operation; E, callous tissue.



Tumor albus of three years' duration in a girl of 6½ years. The destruction of bone tissue is mainly confined to the external half of the epiphysis of the femur and to the adjacent portions of the diaphysis. A, subcutaneous cheesy abscess, communicating with the diseased area of the bone; B, probably a sequestrum surrounded by necrotic tissue; C, shows enlargement and squaring of epiphysis of tibia, as compared with the normal, indicating congestion and proliferation before absorption of the bone tissue has begun.

DIAGNOSIS.—The diagnosis is based mainly on the chronic character of the disease, with the characteristic symptoms of lameness, swelling, and deformity. In the early stages, the bony enlargement which precedes the swelling of the soft parts is very characteristic. The tuberculin reaction is often of aid in diagnosis. Roentgenograms are of great assistance in the diagnosis of the early stages of the disease, as they often show the tuberculous focus in the bone. In differential diagnosis the following conditions must be considered.

Sprains.—The lameness in a sprain of the knee is more acute and of shorter duration. It is not very common in children, and it is wise not to make a diagnosis of sprain, unless there is a definite history of injury, until after recovery has occurred.

Traumatic Synovitis.—This condition, usually spoken of as " water on the knee," is comparatively common in children. It is distinguished from tuberculosis by its acute onset, by the history of trauma, and by the fact that from the beginning examination shows an excess of synovial fluid within the joint.

Infectious Arthritis.—The infectious arthritis which is a part of rheumatic fever, is easily recognized by its acute febrile onset, and by the usual association of evidences of a cardiac lesion. The chronic forms of infectious arthritis are difficult to distinguish from tuberculosis. The enlargement of the bone is absent in chronic infectious arthritis, and the disease usually does not show so typical a combination of the symptoms of lameness, swelling, and deformity. Pain is often more marked in the early stages. In many cases, however a differential diagnosis can only be made by means of the X-ray, which shows a complete absence of changes in the bone.

Scurvy.—Infantile scurvy frequently shows swelling and tenderness near the knee joint. It occurs usually at a much younger age than does tuberculosis of the knee, and careful examination will usually show that the joint is not involved and that there is no limitation of motion at the joint. The rapid response to antiscorbutic treatment quickly clears up the diagnosis.

PROGNOSIS.—While the prognosis as to life is good, tuberculosis of the knee joint is in general that of tuberculous infection at the particular age. It should be stated that tuberculosis of the knee is a less dangerous form than is either tuberculosis of the hip or of the spine. Not only does there appear to be less danger of the development of general tuberculosis, but a fatal ending from prolonged suppuration, amyloid disease and exhaustion, is much more uncommon. The prognosis as to the function of the joint depends upon how early the diagnosis is made and the skill with which the treatment is carried out. With early diagnosis and proper treatment the tuberculous process may be limited to the bone, not involving the soft parts at all, and in such cases there is slight, if any, permanent lameness or deformity. In cases coming under treatment at a later stage, there is a variable amount of permanent stiffness of the joint, lameness, and deformity, depending upon how far the process has progressed and upon how much ankylosis has resulted from the healing process.

TREATMENT.—Patients with any joint disease should be given the benefit of the general treatment of tuberculosis. Local treatment should at the earliest possible moment be placed in the hands of an orthopedic surgeon.

TUBERCULOSIS OF OTHER JOINTS

The symptoms of tuberculosis of other joints, all of which except the ankle are much more rarely involved than are the hip, spine, and knee, bear so close a resemblance to those of tuberculosis of the joints already described, that they do not require any detailed description. The principal features of tuberculous joint disease are always an insidious onset, a chronic course with limitation of motion, pain, swelling, and deformity.

TUBERCULOUS DACTYLITIS

This disease is in reality a tuberculous osteomyelitis, but as it is most frequently seen in the phalanges of the hands and feet, it is most frequently spoken of as tuberculous dactylitis. The process may involve a single phalanx or more than one. It is a chronic tuberculous inflammation occurring in the shaft of the bone, accompanied by enlargement of the bone and thinning of the cortex. In the later stages, there is an inflammation of the periosteum and surrounding soft parts, and the process may go on to necrosis, the formation of abscesses, sinuses, seqestra, etc. The disease is very chronic, lasting from one to three years, and it often results in marked deformity.

While tuberculous dactylitis occurs most often in early childhood, it is seen much more frequently in infants than is tuberculosis of the large joints. At times it is the only manifestation of tuberculosis outside the primary lesion.

SYMPTOMS.—In the majority of cases, the only symptom in the early stages of the disease is a painless enlargement of one or more of the phalanges. The enlargement is very chronic, and it may be several months before the swelling is of sufficient size to attract attention. In exceptional cases there is a more active inflammation, accompanied by pain and tenderness. When the swelling is fully developed the appearance is quite characteristic, there being a spindle-shaped enlargement of the entire phalanx which is smooth, hard, and uniform. In a certain number of cases, the diseased process is arrested before necrosis occurs, and in such cases there is a gradual lessening of the swelling, although usually never complete disappearance. In the majority of cases, however, discoloration of the skin appears and an abscess opens at one side of the finger. The abscess cavity contains a cheesy pus, and may also contain a sequestrum of dead bone. Around the cavity there is a thin layer of new bone formed from the periosteum. In neglected cases the discharge continues for a long time, several abscesses may open, and there may be exfoliation of several sequestra, which may involve the shaft of the entire phalanx. After the process has healed, there is usually shortening and deformity of the finger, and sometimes the disability produced is so extreme that amputation is necessary.

DIAGNOSIS.—The appearance of the fingers or toes in dactylitis is so characteristic that the diagnosis can be made at once from the peculiar fusiform swelling. It must be remembered, however, that there is also a syphilitic dactylitis, in which the appearance of the affected fingers is precisely like that seen in tuberculosis The differential diagnosis depends upon the general evidences which may be present of tuberculous infection on the one hand, and of syphilis on the other. The tuberculin reaction and the Wassermann reaction are of aid. It must be remembered that tuberculous dactylitis is much more common in children than is syphilitic dactylitis. The differential diagnosis may be made immediately and with absolute certainty by means of the X-ray. The plates will show that in tuberculous dactylitis the process involves the shaft of the bone, whereas in syphilitic dactylitis it consists mainly in a thickening of the periosteum. Syphilitic dactylitis is more apt to occur in younger infants and to be multiple and symmetrical, than is tuberculous dactylitis.

PROGNOSIS.—This depends upon how far the process has gone. With abscess and sequestrum formation the prognosis as to a useful finger is rather doubtful. If on the other hand, the nature of the process is recognized during the stage of fusiform swelling, and if thorough treatment of tuberculosis is instituted, the prognosis is very much better.

TREATMENT.—The most important thing is the immediate institution of all the measures used in the treatment of chronic tuberculosis described in Volume I.

As far as local treatment is concerned, the most important measure is the keeping of the diseased part at rest. If a finger is involved, a finger splint should be used Early excision for the purpose of arresting the disease is not to be recommended. When abscesses form, however, they should be opened early and freely, and all sequestra and loose fragments of diseased bone should be removed. The finger must be kept in the proper position, and the wound should be treated on general surgical principles. All such measures as painting with iodin or other local applications are entirely useless.

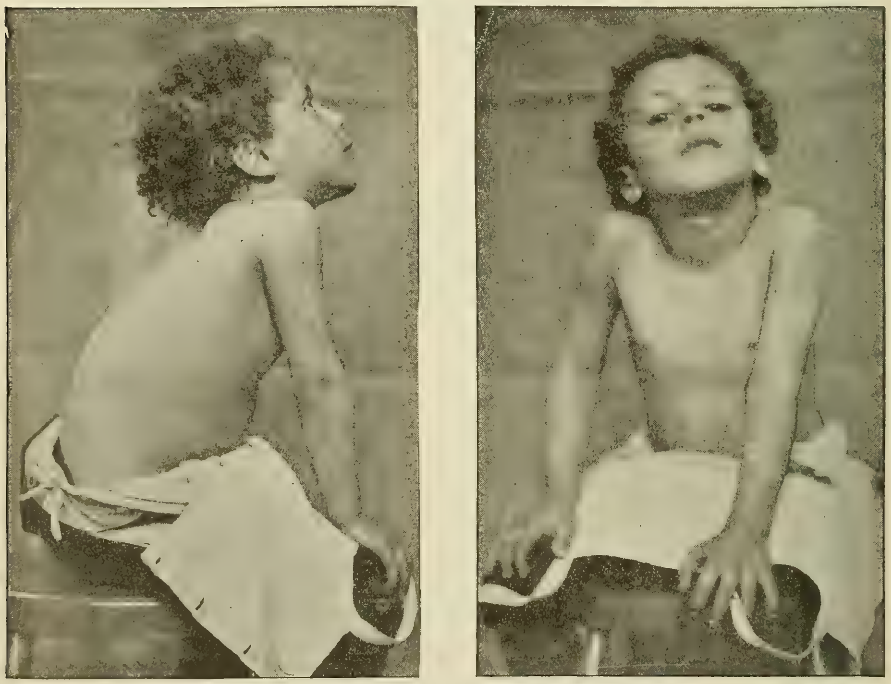
MYALGIA—ACUTE MYOSITIS

Myalgia is an affection characterized by pain and stiffness in certain groups of muscles. It may be acute or chronic.

ETIOLOGY.—The disease is usually seen in children between the ages of five and fifteen, and generally follows exposure to cold or dampness and to bad hygienic surroundings. It has been held by Leube, Peltessohn, and others to be due to infectious microörganisms, but there is as yet no proof of this.

PATHOLOGICAL ANATOMY.—Not enough pathological material is available in this condition to afford a basis for a description of the lesions. It is probable that the muscles are the seat of a true inflammatory reaction with exudation. If this is the case, myositis is a more fitting term than myalgia.

FIG. 259



Acute rheumatic torticollis. Fifth day of attack

SYMPTOMS.—Pain on movement and tenderness and stiffness of certain muscles are the typical symptoms. Fever is rarely present. The pain is at times quite severe, slight movements of the muscles bringing on sharp exacerbations. Certain names have been given according to the group of muscles affected, such as *cervicodynia*, or *acute torticollis*, when the process is located in the muscles of the neck; *pleurodynia* when in the intercostal muscles; *lumbago* when in the lumbar muscles; *cephalodynia* when in the muscles of the head; *scapulodynia* and *omodynia* when in the muscles of the shoulder.

The disease may be acute or chronic, the acute cases recovering in a few days. The chronic cases may persist for weeks, often leading to increase in the connective tissue, producing muscular stiffness and contractures.

DIAGNOSIS.—The diagnosis is to be made from pleurisy by the absence of physical signs and by the pain and muscular tenderness. It is distinguished also from neuritis, in which the tenderness is along the course of the nerves and in which massage is not efficient.

TREATMENT.—Drugs are of less use than hot air and massage. Heat may be applied by means of a hot flat-iron rubbed on the affected part protected by a piece of thick flannel. Massage is especially to be recommended. Electricity is of service in chronic cases. Phenacetine and the salicylates are the drugs which are indicated; they should be used with caution, and only if they seem to benefit the especial case, and should not be employed as a routine treatment.

PROGRESSIVE MUSCULAR DYSTROPHIES

CLASSIFICATION.—The group of diseases known as *primary muscular dystrophies*, or *primary myopathies*, are represented by several forms, which differ from progressive muscular atrophy of central origin (Aran-Duchenne type) in that the lesions are primary in the muscles and are not dependent upon degenerative changes in the spinal cord.

For the sake of convenience Erb has divided the muscular dystrophies into two large groups, which are still further subdivided into types dependent partly on pathological and partly upon clinical differences. This classification may be expressed as follows:

I. Progressive Muscular Dystrophy of Infancy.
    A. Hypertrophic Form, in which the muscles are increased in size, and eventually become atrophic.
        a. With real hypertrophy of the muscle fibers.
        b. With false hypertrophy, the muscular fibers having undergone lipomatosis.
    B. Atrophic Form, in which there is no stage of hypertrophy.
        a. The Landouzy-Dejérine type, in which there is primary involvement of the face.
        b. An atrophic form without involvement of the facial muscles.
II. Progressive Muscular Atrophy of Youth and Adult Life. This is also known as *Erb's juvenile* form.

All these forms have certain points in common. There is a distinct hereditary influence shown by family association of cases. The electrical reactions are quantitatively diminished but not qualitatively altered in all forms, but none shows a typical reaction of degeneration. The reflexes are diminished proportionately to the degree of muscular atrophy. As Sachs has pointed out, the chief distinction between the different clinical types is in reference to the distribution of the atrophy or hypertrophy.

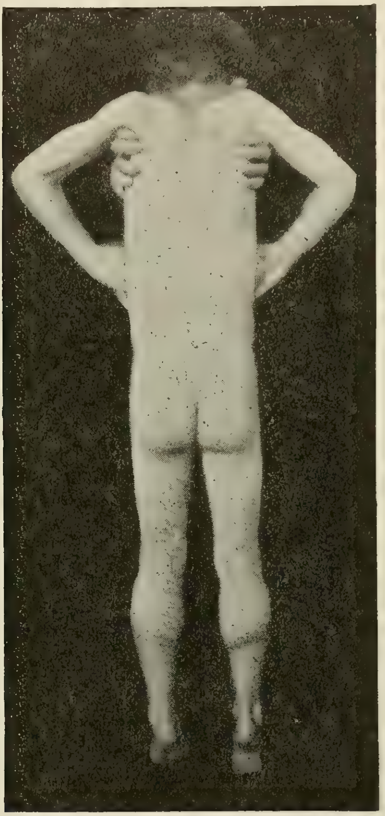
ETIOLOGY OF THE MUSCULAR DYSTROPHIES.—Heredity shown by association of cases in one family is the only etiological factor of any importance, so far as is known. This peculiarity serves as the most important point in the differentiation of the dystrophies from the progressive central muscular atrophy, almost all cases of which, except Erb's infantile form being free from hereditary influences. The disease is usually transmitted through the mother, who may not herself be affected, may run through as many as five generations, and generally attacks the males. The disease, as a rule, begins before puberty, rarely after the twentieth year.

PATHOLOGICAL ANATOMY.—The chief histological changes in the muscular dystrophies consist in hypertrophy and atrophy of the muscular fibers, with proliferation of the nuclei, vacuolization and segmentation of the fibers. The hypertrophy of the muscle fibers appears to be an early process in all forms of muscular dystrophies, and is associated with lipomatosis and changes in the connective tissue. Atrophy of the muscle fibers is a later stage of the disease. No sharp distinction histologically can be drawn between the muscular lesions of the spinal amyotrophies and the muscular dystrophies, as hypertrophy of the muscle fibers may rarely occur in the spinal amyotrophies (Sachs). The dystrophies are, however, not associated with lesions of the central nervous system.

SYMPTOMS. PSEUDOHYPERTROPHIC FORMS.—This disease usually develops in early childhood, from the second to the seventh year, nearly always before the tenth year The earliest symptoms noticed are weakness of the muscles, a waddling, clumsy gait, and awkwardness in the ordinary motions, especially in rising from the floor or climbing stairs. These symptoms generally precede any noticeable enlargement of the muscles. Gradually, however, the muscles of the calves increase in size and become hard and firm. The deltoid, supra-, and infra-spinati muscles are similarly affected. The thighs are rarely hypertrophied. Atrophy of the thighs and of the deep muscles of the back and shoulder sets in. The atrophy of the serratus magnus gives the characteristic "angel-wing scapulae." Late in the disease the atrophic process becomes general, affecting even the hypertrophied calves. The muscles of the face are not involved until the very last stages.

Children who are affected with this form of dystrophy learn to walk late, and assist themselves by leaning on the furniture or other objects in their path. When the child is placed on the floor on its back it has difficulty in getting up. It has to turn over on its face first, and then to aid the weakened muscles of the legs and trunk by means of the hands and arms, climbing up, as it were, upon itself by placing the hands upon the knees and then farther and farther up the thighs. Fibrillary contractions do not occur. The knee-jerks in some cases disappear as the disease advances. Sensation, as a rule, is normal. There is seldom any disturbance of the bladder or rectum.

FIG. 260



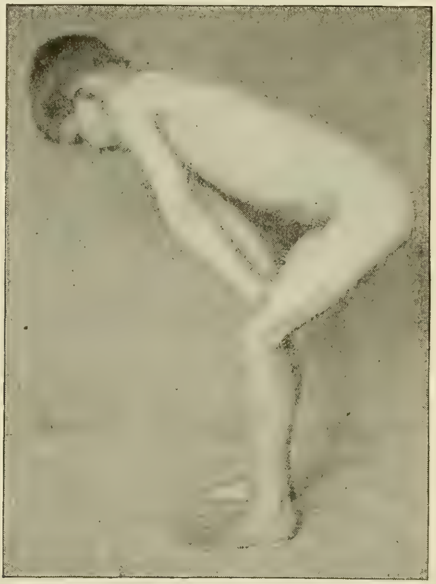
Pseudo-hypertrophic muscular paralysis, showing enlarged calves

In the later stages of the disease contractions of the muscles occur, and in this way permanent distortions of the joints may result. The most common deformities are talipes equinus and flexion of the knees and hips.

Lateral curvature may occur, or a permanent flexion of the spine from weakness of the erector spinae muscles may result, so that the child sits bowed forward, or when kneeling on the hands and feet there is a saddle-shaped depression of the back. The electrical reactions show only quantitative changes. The reaction of degeneration is never present.

ATROPHIC FORM.—This form is known as the Landouzy-Dejérine type. The muscles of the face and shoulder are primarily affected; there is no hypertrophy except in very rare instances. The muscles of the forearm, hands, legs, and back remain unaffected. Westphal has described a case in which the face muscles were involved in an otherwise typical case of pseudohypertrophy. Other forms occur without involvement of the facial muscles. The Landouzy-Dejérine type, therefore, is not absolutely distinctive.

FIG. 261



Pseudo-hypertrophic muscular paralysis, showing position assumed in rising from the floor

ERB'S JUVENILE FORM.—The shoulder muscles are primarily affected. The disease begins in late childhood or early adult life and is characterized by progressive atrophy of the muscles of the shoulder, upper arm, pelvis, thigh, and back, the forearm and legs remaining normal. True or pseudohypertrophy may be associated with the atrophy in certain groups of muscles, notably in the deltoids, supraspinati and infra-spinati muscles.

DIAGNOSIS.—The differential diagnosis of the different types of the progressive muscular dystrophies presents many difficulties, and depends primarily upon the distribution of the lesions.

The group as a whole differs from the spinal or central form, however, in the hereditary character of the affection, in the primary appearance and the distribution of the lesions, in the absence of fibrillary contractions, in the absence of marked electrical changes, and in their greater frequency in early life.

The chief point in the diagnosis from progressive neural muscular atrophy is in the peculiar distribution of the atrophy in the latter affection to the muscles of the feet and to the peroneal group.

PROGNOSIS.—All forms of muscular dystrophies are slowly progressive and are incurable. The pseudohypertrophic type may render the patient a helpless cripple in two or three years, and the patient is very susceptible to intercurrent diseases.

MYOTONIA CONGENITA

(Thomsen's Disease)

This is a disease characterized by a spasmodic inhibition of voluntary movement. It has usually been classified with other spasmodic affections under diseases of the nervous system, but the anatomical evidence suggests that it is a primary myopathy, without any lesion of the nervous system or disturbance of nervous function.

ETIOLOGY.—The only known etiological factor is that of *heredity*. The disease is almost always inherited, usually from a maternal grand-parent, and it may occur in several members of the same family. In almost every case it manifests itself in early childhood.

PATHOLOGICAL ANATOMY.—Lesions are found only in the voluntary muscles. The muscle fibers are markedly increased in size, with a marked increase in the number of nuclei, and a slight increase in the perimysium. There are no evidences of degeneration or atrophy. In the sarcoplasm, however, there are numerous small round colorless or yellowish dots, which suggest that there is an actual disease of the muscle fibers.

SYMPTOMS.—The symptoms are noticed only during voluntary movements, the contraction of the muscles responding very slowly to the will, and persisting for a little time after the individual has willed the muscular movement to cease. After a period of quiescence, the muscles become stiff and respond to the will only with difficulty. On repeated efforts to overcome this difficulty the same movement becomes more and more easily accomplished. A tap upon the muscles causes a slow, tonic contraction, which relaxes slowly. Long-continued rest, heat, cold, and excitement cause an exaggeration of the symptoms. The muscles of the arms and legs are those usually most often implicated. Sensation and the reflexes are normal. The muscles are apparently enlarged, giving at times the appearance of hypertrophy, but the strength of the muscle is not proportionate to its size. Erb has described a characteristic electrical reaction, called the *myotonic reaction*, in which the contractions caused by electrical stimulation of the muscle with either the galvanic or faradic current attain their maximum slowly and relax slowly, and wave-like contractions pass from the cathode to the anode. Electrical stimulation of the nerves causes normal or diminished contraction.

DIAGNOSIS.—The diagnosis is made by the characteristic difficulty in making volitional movements, by the peculiar contraction which follows a slight tap on the muscles, and by the presence of the myotonic reaction.

TREATMENT.—Although at times it may recover temporarily, the disease is incurable, and there is no known treatment which is of much benefit, although active exercise is indicated and is in most cases desirable. The most effective treatment is by means of massage, and training of the muscles with systematic exercises.

MYATONIA CONGENITA

(Amyotonia Congenita; Oppenheim's Disease)

In 1900, Oppenheim described a remarkable disease of early life, in which the chief symptom is a hypotonia or even atonia of the muscles. This is accompanied by a limitation of active movement, varying in degree, which in the most marked cases apparently reaches a complete paralysis, the extremities lying motionless. Although on close observation contractions may be brought out in some of the muscles, they are weak and without locomotor effect.

Since 1900, about fifty cases of this condition have been reported in medical literature. I believe, however, that the disease is commoner than is generally supposed, many cases being unrecognized because of the appearance of the symptoms in early infancy, failure of their correct interpretation, and the tendency toward an early fatal ending. It is certainly a commoner condition than Thomsen's Disease.

ETIOLOGY.—This is wholly unknown.

PATHOLOGICAL ANATOMY.—In seven autopsy records, and two additional examinations of the muscles which I have collected, there is some disagreement as to the character of the lesions. Most of them agree as to the general character of the muscle lesions. The great diminution in the size of certain muscle fibers appears to be the significant lesion The lesions are shown in the illustrations. While there is some disagreement in the records as to lesions of the nervous system, the majority of observers have found no lesions in either the spinal cord or motor nerves.

Fig. 262

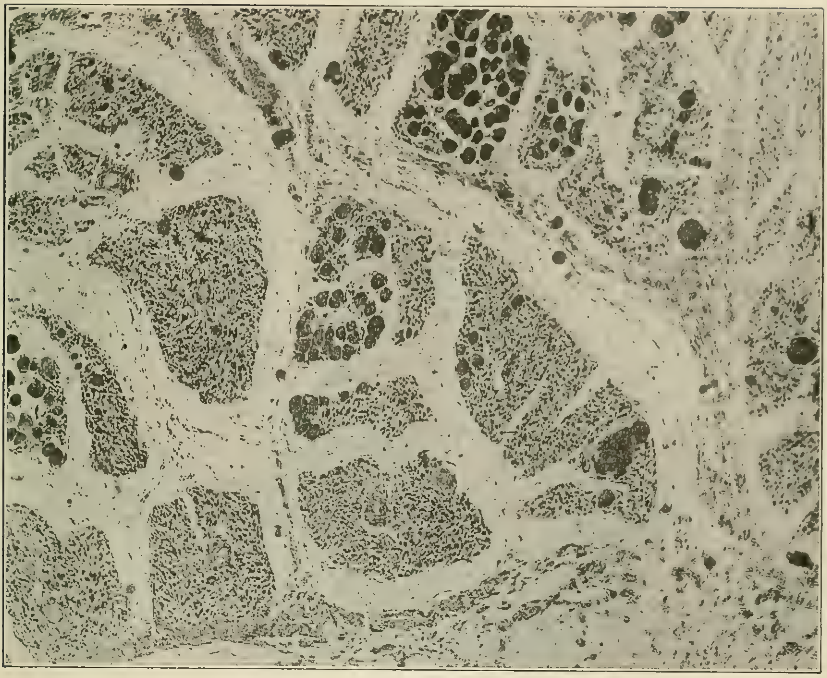


Fig. 263

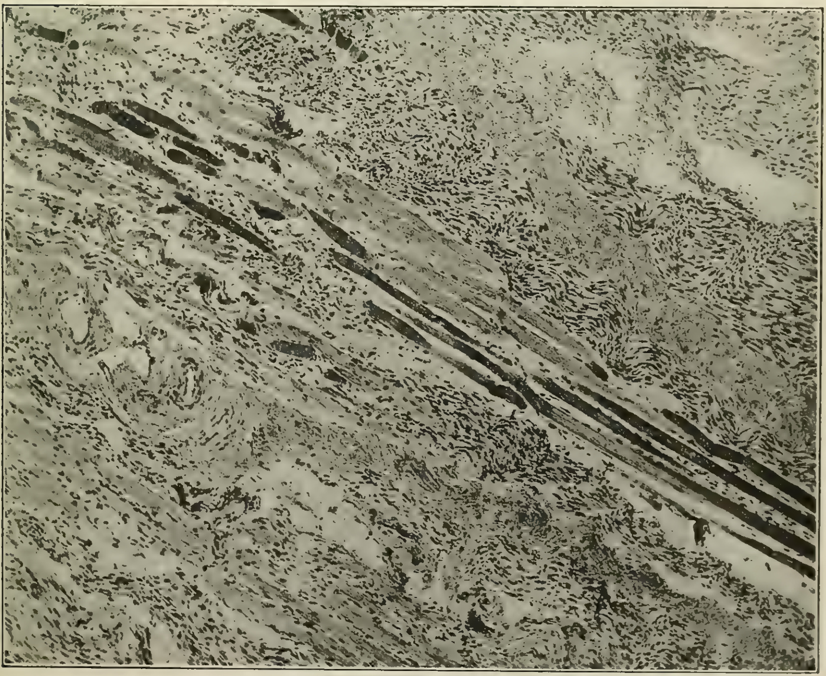


FIG. 264

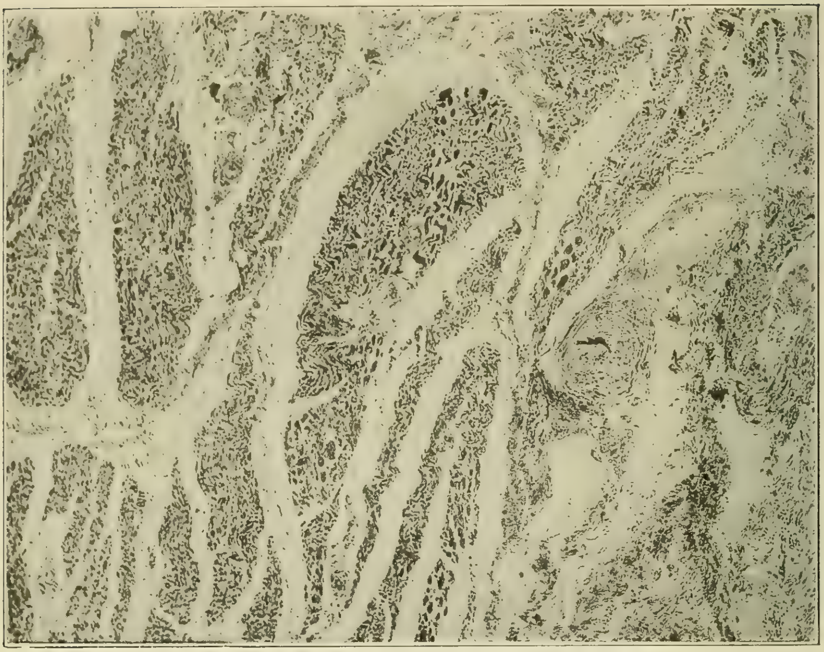


FIG. 265

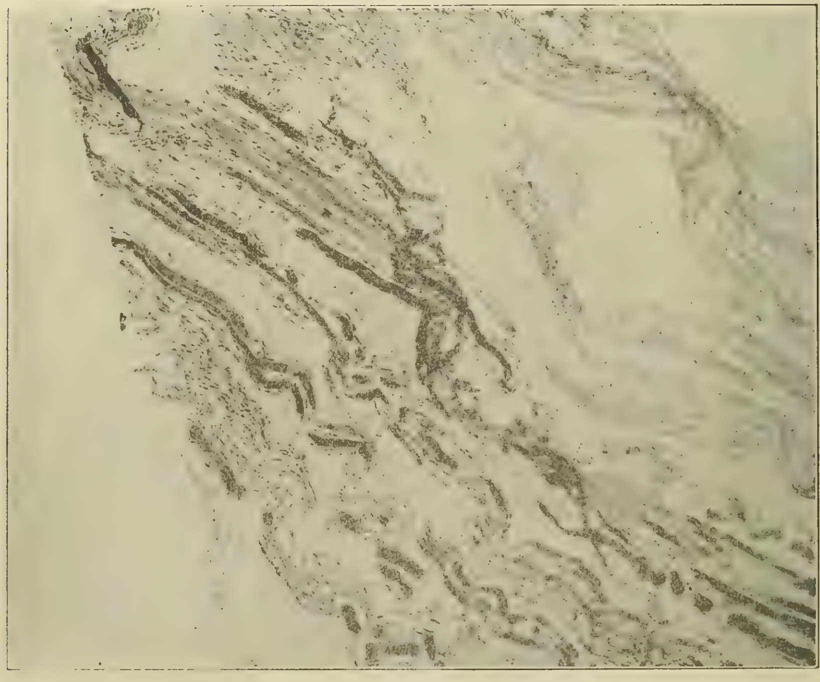


FIG. 266

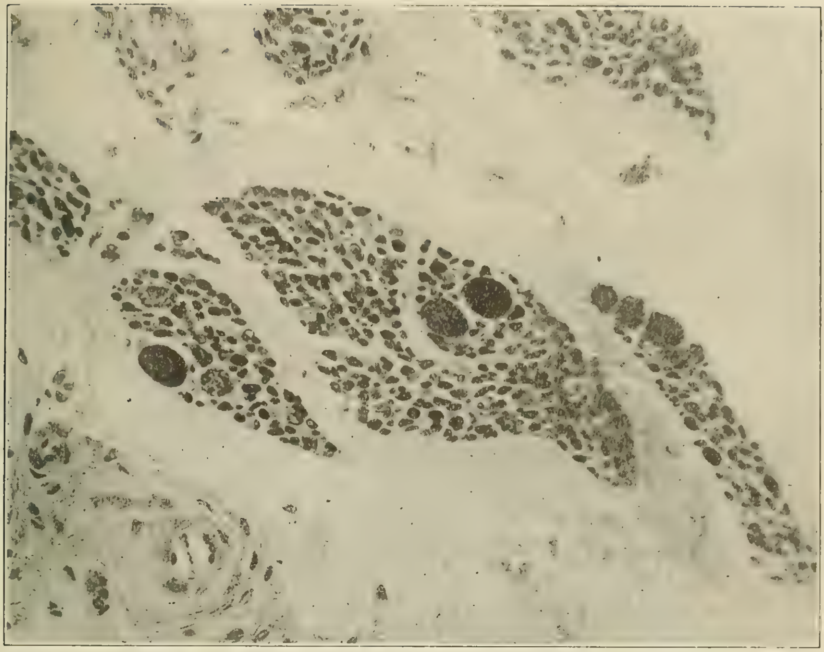
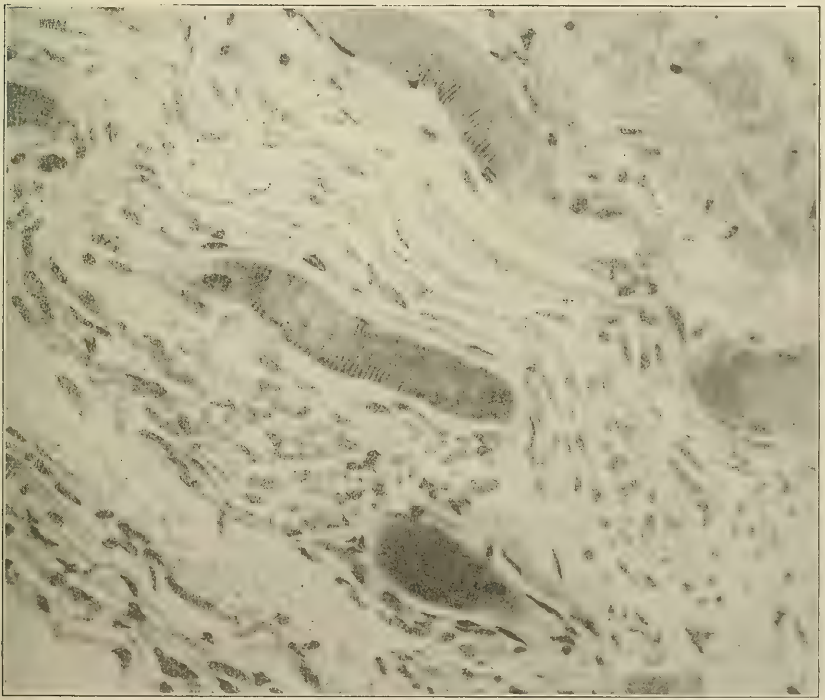


FIG. 267



MYATONIA CONGENITA

FIG. 262—Shows a cross section of the affected muscle, with fasciculi containing a few large, approximately normal fibers, and numerous bodies appearing like cells, but which represent the cut ends of the small fibres.

FIG. 263—Shows the same condition in longitudinal section. There are a few normal fibers, and alongside run numerous small fibers.

FIG. 264—Shows a cross section in which the muscle fasciculi contain only the cell-like bodies representing the degenerate fibers, no normal fibers remaining.

FIG. 265—Shows a longitudinal section in which partly degenerated fibers, midway between the normal and the small fibers are seen.

FIG. 266—Shows a cross section under the high power, in which two large and many small fibers are present in the same fasciculus.

FIG. 267—Shows a longitudinal section under the high power, in which a normal large fiber, with its striations, appears. Beside it run numerous small fibers, in which under the microscope, the striations are plainly visible, though they do not show so well in the photographic reproduction.

SYMPTOMS.—In Oppenheim's cases the lower extremities were always more markedly affected than the upper, and the muscles supplied by the cranial nerves were not affected at all. The muscles of the neck and trunk were affected in one case. In none of his cases were the muscles atrophied, although they were soft. Electrical stimulation showed a varying diminution of contractility up to complete disappearance. There is no disturbance of sensation, and no reaction of degeneration. There is a very marked relaxation of the joints, owing to the flaccidity of the muscles. The affection is congenital, though it may not attract attention for some time after birth. Oppenheim considers the condition to be essentially one of retarded development of the muscles, and that it tends to betterment, although he did not exclude the possibility of involvement of the nerves or spinal cord.

All the main points in symptomatology are covered by Oppenheim's original description. In the reported cases, the extremities are most frequently and most markedly affected, weakness being usually most pronounced in the lower extremities. The muscles supporting the head were involved in quite a number of the severer cases. Most of the reports agree as to the non-involvement of the muscles supplied by the cranial nerves, the only exceptions being two cases in which there was difficulty in deglutition, and one in which there was an inability to close the eyes. The degree of paralysis described in these reports varies greatly. In the most marked cases, no movements could be performed with the muscles affected, except at times feeble movements of the fingers and toes. In some cases contractions were observed in the affected muscles, without locomotor effect. In the milder cases there was no actual paralysis, but movements were feebler than normal, the patients being unable to walk, to stand, to sit up, or to hold up the head.

All the descriptions agree on the great atony of the muscles, and consequent flaccidity of the joints. No description of this can be better than that given by the mother of one of Collier's patients: "By whatever part of the body I held him up, all the rest of him hung down like so many pieces of yarn." Atrophy of the muscles is reported as absent in most of the cases, although in many of them it was stated that the muscles were softer to palpation than normal, with a peculiar doughy feel. There are, however, among the reported cases, a few in which atrophy was noted.

In the majority of cases the lack of movement was noted at birth or very soon afterward. In many of these cases, however, this information as to lack of movement could only be obtained by questioning, the disability not having attracted sufficient attention to be considered important by the mother, or to be a chief cause of complaint. In quite a number of the cases, the condition was noted in the earliest days of life, but only attracted attention much later when it came time for the baby to hold up its head, to sit, to stand, or to walk.

There is considerable variation in the remaining symptomatology as described in the reported cases. Sensation appears to be unimpaired, although an abnormal tolerance of strong Faradic current has been mentioned. The deep reflexes were absent in most of the cases, although in a few of the milder ones, the knee-jerks were reported simply as present, and in a few others, as present but diminished. The reports on the reaction to the Faradic current vary greatly, being sometimes present, sometimes absent, most frequently diminished. These variations correspond in general to the severity of the case, as shown by the amount of muscular disability.

The three cases which have come under my observation agree in their symptomatology with the other reported cases. One or two points were notable, however. In all three cases a very striking symptom was the labored breathing of a wholly diaphragmatic type, which could not fail to attract immediate attention. This was due to the paralysis of the intercostal and other accessory muscles of respiration. Labored breathing, without detailed description, is mentioned in only one or two of the cases from the literature.

Another striking feature was the extreme deformity of the chest, which was present in two of my cases. It resembled an exaggeration of the most extreme deformity which is seen in rickets, and was undoubtedly caused by the early paralysis of the accessory muscles of respiration, together with the pull of the uninvolved diaphragm.

A prominent symptom in all three of my cases was the accumulation of secretion in the mouth and throat. This was undoubtedly due to involvement of the muscles of deglutition. In all three cases there was difficulty in swallowing milk, and one baby became utterly unable to swallow, having to be tube-fed from the time it entered the hospital until its death. In all three cases choking attacks were frequent, and one case died in such an attack, while the other two cases died of bronchopneumonia, probably due to inhalation of secretion. Difficulty in swallowing is mentioned in but two of the other reports which I have read. In my cases the involvement of the muscles of deglutition appeared to be a later symptom, and to be progressive.

COURSE AND PROGNOSIS.—In two of my cases a gain in the power of movement was noted. In Case 1 this improvement was temporary only, disappearing later. In Case 2 the slight gain in the power of moving the left arm continued. No tendency toward improvement was ever noted in Case 3. On the other hand, the difficulty in swallowing became worse in all three cases, and in Case 3 the baby lost the power of moving the head from side to side.

In most of the other reported cases, the course of the disease and final outcome were not reported. The cases are by no means confined to infancy, there being 22 cases which were observed in the first two years of life and 18 in later childhood, the oldest being 10 years. In these older children there was a great variation in the amount of muscular weakness, some children being able to sit, stand, or walk. Walking was generally acquired much later than normal. It is not possible in most cases to gather from the reports whether these cases represent the result of improvement in the original condition, or a milder involvement from the start. They only show that there is great variation in the severity of the disease.

In but 24 of the cases is there any report on the progress or outcome of the condition. In 11 cases some improvement, apparently at least, was noted. In two cases the condition was apparently stationary over a fairly prolonged period of observation. In two cases the condition appeared to become progressively worse; nine cases died. It is difficult, however, in early life, to judge whether improvement is real or only apparent. The developing child presents no normal standard, and there is a constant normal gain in the function of movement, which might easily give a false impression of improvement. In other words, because a child with a widespread muscular lesion eventually learns to walk, it is no proof of even functional improvement in muscular power. Then, errors of observation, so important in early life, must be considered.

Nevertheless, there seems to be at least some evidence of a tendency toward improvement. Whether this improvement is only functional, or whether there is any tendency toward improvement in the actual lesion, cannot be decided. Without actual destruction of nerve cells, there should be considerable capacity for regeneration. We do not know, however, whether muscles so altered are capable of complete regeneration, nor of how much regeneration they are capable, if at all. An autopsy on a case which died from some intercurrent affection after showing an apparent improvement in the muscular condition, might give us a valuable addition to our knowledge on this point.

The cases reported up to 10 years of age do not give a very favorable prognosis. In no case has complete recovery been reported. On the other hand, the severer cases appear to tend toward a fatal ending, especially during infancy. Bronchopneumonia is the commonest immediate cause of death Whether in these cases the process may be progressive, or whether the fatal ending is due to the combination of a stationary anatomical condition, combined with the increased demands of the growing child, we do not know.

PROBLEMS AND RESEARCH.—We know practically nothing of the etiology of this condition. The onset appears to be gradual, as the descriptions usually do not give a very exact time of onset. While it appears to be congenital, we cannot even be sure of this, as in those cases in which the condition is reported as having been observed at birth, we cannot be absolutely sure of the accuracy of the observation. Usually the information is obtained at a later period, when parents, in their inability to fix an exact time of onset, are extremely apt to say " ever since birth." Still, it seems to me that the weight of evidence points toward the congenital nature of the disease.

The presence or absence of fetal movements might have an important bearing on the question of the congenital nature of the disease, although the degree to which these are felt presents normal variations, and their absence does not necessarily denote fetal paralysis. In the collected cases, there was mention of fetal movements in twelve. The movements were felt in eight and not felt in four of the cases. In two of the cases in which they were felt, they were reported as less lively than normal, as in two of my cases.

There is no reason to believe that the disease is infectious. The onset, the distribution of the lesions, the absence of focal lesions, or of any lesions pointing toward a past infection, are all against such a supposition. The disease has never been seen in either an epidemic or an endemic form. In none of the cases has the disease even appeared in connection with any acute infection.

It seems much more probable that the disease is toxic, rather than infectious. The action of the toxin appears to be directly on the muscle fibers, or possibly upon the terminal nerves or nerve endings. Councilman believes it probable that such a toxin is produced, not in the infant, but in the mother. There does not seem to be any relation between the disease and any other disturbance in the child. Councilman in our report of Case 1, advanced the hypothesis that there might be a toxin in the blood of the mother, from which she would be protected by the presence of antibodies. Both toxin and antibodies would, during the intrauterine life of the child, be transmitted through the placental circulation. After the birth of the child, the action of the toxin would no longer be counteracted by antibodies received from the mother. Councilman says, however, that under such an hypothesis, it is difficult to explain why, as in our case, single muscle fasciculi and single fibers should remain normal.

I believe myatonia congenita is possibly much commoner than is generally supposed. Two of my cases had been long under the observation of physicians, without any suspicion that there was anything more wrong than weakness and malnutrition. The milder types of the disease, which lived long enough to show obvious involvement of the normal motor functions, would not be so likely to be overlooked. But I believe it possible that babies have been certified as dying of marasmus and bronchopneumonia, who really had myatonia congenita.

DIVISION XV

DISEASES OF THE SKIN

There are no lesions of the skin peculiar to the early years of life. All the diseases seen in adults may occasionally occur in children, although many of them are very rare. The diseases of the skin would call for no attention in a work on disease in early life, were it not for the fact that the lesions of the skin are often modified at that age, and do not always resemble the lesions of the same disease as seen in adults. Another reason for reviewing the subject lies in the fact that certain diseases are relatively much more common in early life. The delicate skin seen in infancy and childhood is very easily affected by certain causes, especially by irritation from without, so that dermal lesions are apt to occur from causes which might produce no lesion in adults. Finally, it is in early life that the eruptive fevers are especially common. Every practitioner has doubtless been struck by the similarity which at times is seen in the cutaneous lesions of the various forms of erythema to such diseases as syphilis, scarlet fever, and erysipelas. It is very important, therefore, to have a fair, general knowledge of the local diseases of the skin as they appear in children, in order that a correct differential diagnosis can be made from the constitutional diseases with dermal lesions, which have to be treated by those who practise among children.

Only those diseases of the skin will be considered which are sufficiently common to be encountered in the course of ordinary practice among infants and children.

The diseases of the skin cannot be easily classified upon an etiologic basis, because the only prominent clinical feature of the disease is the lesion. The character of the lesion depends upon the pathologic anatomy of the skin, and the same lesion may be produced by several causes. Also, in many conditions, several entirely distinct causes coöperate to produce the same disease, while in other diseases, the cause is obscure or entirely unknown. The same causes play a part in producing diseases of the skin as in other diseases We find developmental causes, traumatic causes, toxic causes, infection, and functional disturbance from multiple causes. What we fail to find is any close correspondence between cause and clinical manifestation.

There are a certain number of etiologic factors which play an important part in the cutaneous manifestations of early life, and are responsible for the prevalence of skin disease in infancy and childhood.

The first of these is the delicacy of the skin in infancy and childhood. This makes the skin more vulnerable to all sorts of external agencies, and is not an individual peculiarity, but is common to all infants and children. This factor explains the tendency of many skin diseases in childhood to assume a more acute and inflammatory type than in adults.

The second etiologic factor of importance is a congenital vulnerability, or abnormality of the skin peculiar to some individual infants. Such a condition must be invoked to explain the occurrence of certain cutaneous affections in some infants, and their absence in others, other etiologic factors being apparently the same.

A third etiologic factor is the instability of digestion, metabolism, and elimination in early life. Disturbances easily lead to the formation of toxins, which may act upon the skin either as predisposing or as exciting causes.

A fourth etiologic factor is the excitability and instability of the nervous system characteristic of early life. Nervous influences probably have a greater connection with the vulnerability of the skin than is generally appreciated.

Finally, the fifth etiologic factor, operative mainly in older children, is the public school There is more intimate and promiscuous contact in childhood than at any other age, and the public school is the great disseminator of the contagious parasitic diseases so common at this age.

The causes of the diseases of the skin in childhood may be classified somewhat as follows:

1. Congenital Factors
    a. Normal delicacy of the skin in early life.
    b. Individually peculiar vulnerability, (Constitutional predisposition).
    c. Developmental lesions of the skin, (as in Ichthyosis).
2. Internal Factors
    a. Toxemia from faulty metabolism.
    b. Toxemia from definite foods or drugs.
    c. Toxemia from infections.
    d. Reflex nervous disturbances.
3. External Factors
    a. Mechanical, chemical, or thermal injury.
    b. Parasites.

The various cutaneous affections common to childhood cannot be classified and grouped according to this etiologic scheme, because a number of etiologic factors are usually found in a single diseased condition. They will be considered here under four groups.

1. Developmental conditions. This will include only ichthyosis.
2. Conditions in which external mechanical, chemical, or thermal injuries play a part. This group will include Miliaria, Eczema, and Dermatitis.
3. Conditions due to internal factors,—the so-called toxic affections.
4. Conditions due to infection,—the so-called parasitic affections.

I. DEVELOPMENTAL LESIONS

ICHTHYOSIS

ETIOLOGY.—Ichthyosis is a congenital disease, although in the common mild type, the lesions are not seen at birth, but usually appear at some time in the first two years. The cause is a developmental defect, which disturbs the nutrition of the skin, and the functions of the sweat and sebaceous glands.

SYMPTOMS —The disease is discovered at some time in the first two years of life, and usually begins in the early weeks after birth. Its course is essentially chronic, and it tends to increase in severity during childhood, remaining stationary after adult life is reached. It varies greatly in severity. In the mildest form there is only an abnormal dryness of the skin, which is harsh, and has more prominent furrows than normal. There may be slight scaliness in mild cases. Severer cases show many reticulated scales, which may be small and thin, or larger and thick like fish scales. Especially on the extremities are often seen polygonal scaly plates, bounded by the natural furrows. The whole body is involved.

The disease is better in summer and worse in winter. Itching is often present. There are no inflammatory manifestations.

FETAL ICHTHYOSIS.—This is a severe form of the disease, which is present at birth and is developed probably about the fourth month of intra-uterine life. It is characterized at the time of birth by the existence all over the body of horny epidermic plates separated from one another by fissures and furrows, associated with deformities of the mouth, nose, eyes, lips, and limbs, and leading within a few days or even hours to the death of the infant. The disease does not seem to affect especially the size and weight of the infant. As a rule, the viscera at the post-mortem show nothing abnormal except an unusual degree of congestion. The microscopic examination shows no extension of the keratinizing process on any of the mucous membranes.

In the early hours of life infants with this disease usually cry loudly and continuously, but sometimes the cry is feeble and often very peculiar. The respiration is usually impeded by the blocking of the nostrils with epidermal masses. Suction is rendered difficult or altogether impossible by the presence of ichthyotic plates around the mouth. These infants are, however, usually able to swallow readily. As a rule, nothing abnormal is found in connection with the urine or the feces. Insomnia is a marked symptom.

These infants have a very repulsive appearance, and there is a cadaveric odor arising from the abnormal condition of the skin. This ichthyotic condition of the skin is usually universal, but is most evident upon the face. The mouth is ordinarily kept open by the contraction of the surrounding parts, and from its angles radiate fissures, which simulate the rhagades of syphilis. The lips are thick and everted, so as to form an irregular entrance to the gaping buccal cavity. The chin is receding. The nose can scarcely be seen, as it is covered so thickly with the epidermal plates around the nostrils. There is usually ectropion of both eyelids, but sometimes only of the upper one, the orbits seeming to be occupied by fleshy tumors. If, however, we separate the swollen eyelids, the normal eyeball is found to lie beneath. The external ear seems to have disappeared almost entirely.

In contradistinction to the opinion formerly held that fetal ichthyosis was a general seborrhea, it is now generally supposed to be connected with the disease as it occurs in the adult.

PROGNOSIS.—The severe fetal form is almost invariably fatal. The prognosis as to the common mild form is good as to life and development, but unfavorable as to cure. Relief can be obtained by treatment.

TREATMENT.—External treatment must be relied upon. Very frequent warm baths, followed by the inunction of some oily substance are indicated. I prefer cocoa-butter for these inunctions, but, other oils, such as lanolin, petrolatum, adeps, olive oil, and so forth, may be tried. A certain amount of improvement may be expected.

II. DISEASES DUE TO EXTERNAL INJURIES

MILIARIA

(Prickly Heat; Lichen Tropicus; Red Gum; Strophulus)

Miliaria is a mild inflammatory affection characterized by thickly-set discrete, small papules or vesicles at the mouths of the sweat ducts.

ETIOLOGY.—The cause is excessive perspiration. In children the usual cause is excessive clothing, or very hot weather. The disease is especially common in infants. There is a possibility that gastro-intestinal disease with absorption of toxic products may play a part in some cases, seen in winter, through the elimination of irritating substances through the sweat glands.

SYMPTOMS.—There is a sensation of itching and burning. Over parts of the body, more or less widely distributed, is a rash, which appears rapidly, without constitutional disturbance. The rash consists of very numerous discrete reddish papules, of a size varying from pin-point to pin-head. The papules are surrounded by a reddish areola, and to palpation cause the skin to feel rough and prickly, as if the papules were sharp pointed. On the summits of many papules there may be minute vesicles, containing a clear fluid. These vesicles do not rupture. When there is a very profuse eruption, the zones around the lesions give the skin a generalized redness. The eruption comes in crops, which are usually repeated, if the cause is not removed.

DIAGNOSIS.—I have seen miliaria mistaken for scarlet fever. The vesicles and rough prickly character of the rash should enable one to make a diagnosis. Miliaria has also been mistaken for eczema but is distinguished by the sudden profuse character of the eruption, by the absence of discharge from the vesicles, and by the rapid improvement under a change of weather conditions.

TREATMENT.—The most important part of the treatment is prophylactic. On hot summer days, children should be lightly clad, and should be kept in cool places. Constipation should be relieved, and intestinal disturbance guarded against.

The local treatment consists in bathing, and the use of dusting powder. I use for bathing cool water, with bicarbonate of soda, one tablespoon to the gallon. This is just as effective as the more complex alkaline lotions, but any soothing, cooling lotion gives relief.

After bathing, the parts should be dusted with any good dusting powder; such as—

Menthol....................	gr. x
Acid borici....................	ʒ ii
Talci Venet....................	℥ ii —M

ECZEMA

Eczema is a non-contagious inflammatory disease of the skin, having an acute, subacute, or chronic course, and accompanied by itching and burning.

ETIOLOGY.—Two principal kinds of exciting causes are associated with eczema; the first kind is external, and includes every variety of irritation of the skin from without; the second group is internal, and includes a number of conditions in which the vulnerability of the skin,—its susceptibility to irritation,—is increased by toxic substances acting from within.

The external causes are mechanical, chemical, or thermal irritants, or a combination of these. Among the most important mechanical irritants are scratching in any condition producing itching, excessive friction, irritating clothing, parasites, and any rough handling of the skin. Among the chemical irritants are excessive or abnormal perspiration, acid urine, irritating discharges from the intestines, irritating soaps, and the application of various medicinal agents. Among thermal irritants are exposure to heat and cold. In infants, the two commonest external causes are in my experience, excessive perspiration and scratching.

Among the internal causes associated with eczema, alimentary disorders play the chief part. It may be seen in association with any form of digestive disturbance, but is especially common in those forms of indigestion which are produced by general overfeeding, by a quantity of fat in the diet too high for the digestive and assimilative powers, or by indigestion from carbohydrate. General overfeeding accounts for the many cases seen in well nourished babies, who have few symptoms of indigestion. Many breast-fed babies with eczema will show excessive fat in the stools, and a very high percentage of fat will be found in the mother's milk. While eczema is occasionally associated with the sugar indigestion seen in the first year, it is most particularly associated with too early or excessive use of starch.

Among other internal conditions with which eczema is associated are constipation, deficient elimination through the kidneys, food idiosyncrasy, and a condition characterized by scanty concentrated urine, seen in children who take an excess of solid food. Eczema is occasionally associated with such conditions as dentition or general irritation, in which the connection with eczema must be explained by reflex action. There is also evidence that at times hereditary influences play an important part. Eczema is frequently seen in children of gouty or neurotic antecedents.

The evidence of causal connection between eczema, and the great variety of conditions with which it is associated, is not always conclusive. In many conditions there is no evidence beyond frequency of association; in other conditions, in addition to frequency of association, we have evidence of clinical benefit when the suspected cause is removed. Evidence of this character is only suggestive, never conclusive. In the case of the external irritants, the evidence is fairly conclusive that any one of them can produce eczema. In the case of the internal causes, a positive etiologic connection between eczema and the gastro-intestinal disturbances associated with it, has been most conclusively proven, in some cases. The digestive balance has been found to be so delicate, that the eczematous lesions may be made to come and go with changes in the diet, with almost the accuracy of an experiment.

It is quite easy to comprehend how the external irritants can produce an inflammatory disease of the skin, such as is seen in eczema. But the same irritants do not always produce the disease, and the occurrence of eczema in some individuals, and its absence in others, must be explained by other factors. Just how the internal causes act has never been proven, and the explanation is theoretical. The generally accepted explanation is that the internal causes produce a toxemia, and that the toxins circulating in the blood increase the irritability of the skin, and the tendency to inflammatory reaction. In gastro-intestinal affections associated with eczema, it is not known whether the toxins are absorbed from the intestinal tract, or are formed in the system as products of the disturbed general metabolism which we now believe to be a part of many digestive disturbances. The result would be the same in either case. Most of the internal conditions associated with eczema are of such a nature that the association may be explained on the basis of the toxic products of disturbed metabolism. The occurrence of eczema in patients of gouty heredity can be explained in the same way.

Whether the toxins act directly on the skin as a tissue, or whether they act upon the skin through the medium of the nervous (vaso-motor) system, or whether by both modes of action, we do not know. It is evident in any case that they create a cutaneous weakness or vulnerability. The fact that among the internal causes are numbered reflex irritation and neurotic inheritance, suggest that action on the skin through the vaso-motor system does actually play a part.

Again, it is not known whether the internal causes *alone*, without any external irritation, can produce eczema. Cases are seen, in which there is no apparent external irritation. But the skin, coming in contact with the outside world, is normally and continually subjected to stimuli which may be considered as relatively irritating. It is best to consider eczema as the result of the joint action of external and internal causes. In a baby with a chronic disturbance of metabolism, increased vulnerability of the skin is constantly present, and eczema appears when an external irritant is added. The greater the constitutional factor, the less is the external irritation required. A very severe external factor may excite the disease in a skin of relatively normal vulnerability. Conversely, normal contact with the outside world may be regarded as a constantly present relative irritation, insufficient to produce eczema, until some sudden disturbance of metabolism, as in an attack of indigestion, intervenes, and appears to be the immediate exciting cause of the attack.

It is difficult to comprehend how so definite a pathologic entity as eczema can be the direct result of so great a diversity of causes. The fact is significant that in many children, both the internal and external causes usually associated with eczema may be present to a marked extent, and yet the skin disease does not develop. On the other hand, many severe and resistant cases of eczema are seen in which very slight causes are found. These facts suggest the possibility of an underlying constitutional cause, which is present in some children and absent in others. Such a cause corresponds closely to Czerny's "Exudative Diathesis." We may not be prepared to agree with Czerny's theory to its full extent, but nevertheless the phenomena of the occurrence of eczema strongly suggest the possibility of some fundamental underlying cause. Under this theory we must conceive that some individuals possess, as a constitutional peculiarity, skins which have an abnormally strong tendency to react in a certain way to a variety of causes. The constitutional peculiarity is situated in the skin itself, and consists of an increased vulnerability, of which eczema is the characteristic symptom, appearing when various predisposing and exciting causes are present.

Another etiologic factor, which accounts for the great frequency of occurrence of eczema in infancy, is the extreme delicacy of the skin, seen at that age, and its more intense glandular activity. The most susceptible age is the first twelve months of life; it is rare for the common infantile types of eczema to develop after this period, although cases developing in the first year, may occasionally persist through the second, or even through the third years.

Eczema may be associated with malnutrition, but rarely. It is commonest in fat babies of healthy appearance.

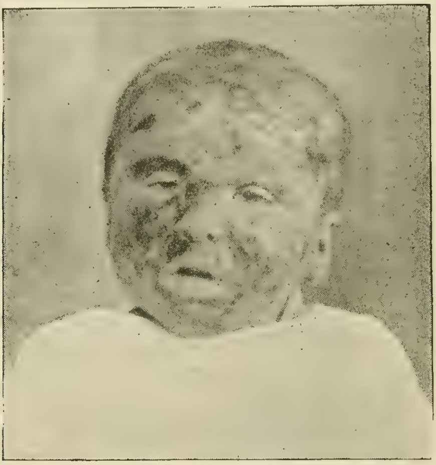
In the secondary lesions of eczema, it is probable that an important part is played by infecting microörganisms.

SYMPTOMS.—Older children are occasionally liable to any of the varieties of eczema seen in adults. There are three types which are particularly common in infancy. These are, Eczema Rubrum, Intertrigo, and Subacute or Chronic Papular Eczema.

ECZEMA RUBRUM (VESICULAR OR PUSTULAR ECZEMA OF THE FACE) (ACUTE EXUDATIVE ECZEMA.)—This is the commonest type seen in early life, and usually appears during the first year. It occurs most commonly upon the face, but may occur on any part of the body. On the face, the cheeks and forehead are particularly affected, though the eruption may extend first to the ears, neck, and scalp, and finally even to the trunk and extremities.

The eruption is characterized by redness, swelling, infiltration, exudation upon the surface, and finally, in many cases, by crusting. Small papules appear, usually on the cheeks, and soon coalesce, forming a red, raw surface from which serum exudes. The discharge may become sero-purulent. It dries, forming yellowish or brownish crusts. Itching is intense, and when the scratching produces bleeding, the crusts are colored by the dried blood. When the crusts are removed, a red, raw, weeping surface is disclosed, which bleeds easily. The intensity of the process varies greatly both in different individuals, and from time to time in the same individual. In mild cases, or in the remissions frequently seen in severe cases, the skin may show only redness, induration, and scaling, without external exudation, and the condition is the same when the disease spreads to the trunk or extremities. Crusts are rare except on the face. The lymphnodes near the eruption are usually swollen.

FIG. 268



Eczema capitis

Infants with this type of eczema are usually well nourished, if not decidedly fat. Nutrition may appear to be unimpaired for a considerable time, but in many cases, the itching and discomfort cause nervous symptoms and loss of sleep, which finally may seriously affect nutrition.

A pustular eczema, or impetiginous eczema is seen when the lesions of vesicular eczema become infected with pus-forming micrococci, although it may develop as such. This form is less common in infants than in poorly nourished children from two to six years of age. It is commonest on the scalp, though it may be seen on the face. Rupture of the pustules is followed by the formation of greenish-yellow crusts, matting the hair of the scalp. They are often very numerous, but are usually discrete, though they may coalesce to form large crusts. There is little itching in this form. The cervical lymph nodes are enlarged. Continual reinfection makes this disease obstinate, and it is probable that it may be communicated to other children.

SUBACUTE OR CHRONIC PAPULAR ECZEMA.—This is the type which in infancy and early life stands next in frequency to eczema rubrum. It is characterized by the formation of patches of varying size, and very irregular distribution. The primary seat of the eruption is usually on some part of the trunk or extremities, and in some cases it remains limited to a single part, or remains unilateral. In most cases there is irregular and disorderly spreading.

In typical cases, the areas are of a pale-red color, exfoliate freely, and are covered with a varying number of pin-head-sized, round or acuminate, reddish elevations, either discrete or closely aggregated. Sometimes some papules may be seen to be surmounted by very minute vesicles. A few widely separated, discrete papules are usually found in the vicinity of the patches.

Itching is very intense in this variety of eczema. It occurs most commonly in infants, but is seen in older children more often than the vesicular eczema of the face.

INTERTRIGO. (ERYTHEMA INTERTRIGO). (ECZEMA INTERTRIGO).—This condition is primarily not a variety of eczema, but is a form of traumatic erythema occurring chiefly in those regions where skin surfaces are in apposition. Moisture and want of cleanliness are contributing causes. It is seen in the regions of the groin, about the arms and buttocks, in the axillae, and about the neck. In its simple, initial stage there is a simple erythema, which may become an intense uniform redness. Under conditions of continued irritation, a true eczema develops, the skin becomes excoriated, leaving a moist surface with a mucoid discharge. There is a feeling of heat and soreness, but no itching and no crusting. The condition of the skin in a fully developed case of intertrigo much resembles that seen in a superficial burn.

DIAGNOSIS.—Eczema in general may be recognized by its cardinal manifestations. These are redness; the development of papules, vesicles, or pustules; the appearance of mucilaginous surface discharge; the presence of crusting or scaling; thickening or infiltration of the skin; itching and burning. Not all of these are usually present in an individual case, but their grouping is usually sufficiently characteristic to permit diagnosis.

The common eczema of the face and scalp seen in infants is so typical as to present no difficulty in diagnosis. Some types, especially those seen on the body and extremities, may resemble the lesions found in other conditions.

Scabies is the disease most frequently confounded with eczema. It is recognized in the first place by the characteristic distribution of its lesions, which are found mainly in the webs of the fingers, on the flexures of the wrists and elbows, in the folds of the axillae, and about the nipples, umbilicus, buttocks, penis, and the insides of the thighs and legs. The itching of scabies is apt to be worse at night. Other members of the household are apt to be simultaneously affected. The diagnosis of scabies may usually be confirmed by finding between the fingers or on the wrists, the characteristic burrows of this disease.

Syphilis is so little inflammatory in its manifestations, as to be not often confounded with eczema. Patches of papular eczema when near the anus may resemble condylomata, but the failure to find any other syphilitic manifestations usually suggests the correct diagnosis

Dermatitis from mechanical, chemical, or thermal irritants may so closely resemble eczema as to be indistinguishable. It is a question whether the distinction usually made by dermatologists between eczema and dermatitis is warranted. In general, the lesions of dermatitis develop more acutely, are characterized by burning rather than itching, and are more amenable to treatment. There is usually more swelling in dermatitis, and often the myriads of minute closely aggregated vesicles, without crusting, give the clue to the diagnosis.

Herpes Zoster presents a vesicular eruption, which is to be distinguished from eczema by its unilateral distribution, tending to follow the area supplied by a nerve trunk; by the large vesicles, their arrangement in clusters, and the lack of tendency to spontaneous rupture; finally, by the presence of neuralgic pain, instead of itching.

Impetigo Contagiosa is closely simulated by pustular eczema. Both conditions represent an infection with pyogenic cocci. In impetigo contagiosa the disease does not begin with aggregated pustules, as in eczema, but begins with discrete vesicles or blebs, which are more superficial, and rupture rapidly, forming yellow " stuck on " crusts.

Erysipelas, has been taken for eczema, but the resemblance is very superficial. It is easily recognized by the fever and constitutional symptoms.

PROGNOSIS.—There is almost no tendency toward spontaneous improvement or recovery during the first year of life. Toward the end of the first year, such a tendency develops, and many cases disappear for good at this period. Other cases improve at the end of the first year, but many have relapses later.

To obtain improvement during the first year, patient and persistent treatment is required. The response to treatment varies very much with individuals, as would be expected from the complex etiology of the disease. Some cases react favorably to local treatment, others to constitutional or dietary treatment, while still others are resistant to all forms of treatment. In a given case, prediction is difficult. Usually several months of treatment at least are required, the prognosis depending mainly on how well the treatment is carried out. The prognosis is also affected by the severity of the case, and the length of time that the disease has existed. Intertrigo is usually easily cured, unless the patients are very poorly nourished from prolonged gastro-intestinal disturbance.

TREATMENT.—In eczema the first step in treatment is the finding and removing of all possible causes. When any error in diet or feeding is manifestly present, it should be corrected. All possible local causes of external irritation, and of reflex irritation, should be removed. A combination of general and local measures is necessary, to obtain good results.

GENERAL TREATMENT.—As disturbances of digestion and assimilation are the most frequent associated causes of eczema, this function should be the first inquired into. The diet should be investigated, both as to quantity and quality of food.

In breast-fed infants, an analysis of the mother's milk, and of the infant's stools is essential. Often the milk is found to contain too high a percentage of fat, and excessive fat is found as soaps in the stools. If the mother's milk has an excess of fat, or if excessive fat elimination is found by microchemical examination of the infant's stools, the breast milk should be diluted by giving the baby one or two ounces of boiled water with two or three grains of bicarbonate of soda immediately before each nursing. The mother's life and hygiene are to be regulated according to the principles given under breast-feeding. The daily quantity of food taken each twenty-four hours by the infant should also be investigated by weighing the baby before and after each nursing. The quantity of food taken may be reduced by lengthening the nursing intervals, or shortening the time when the child is on the breast, or both. Lengthening the intervals is particularly useful. Some cases of eczema will clear up, if the baby is weaned from the breast, but by no means all. If the child is thriving in other ways, I do not believe that weaning from the breast should be countenanced, unless every variety of general and of local treatment has been exhausted.

In bottle-fed infants, if any disturbance of digestion be found, it should be treated by changes in the diet directed at curing the symptoms. The treatment of such conditions is described under artificial feeding.

The most frequent form of digestive disturbance found is inability to digest and assimilate fat, particularly the type in which there are no pronounced symptoms of indigestion. The condition is recognized by the microchemical examination of the stools, which is an essential proceeding in the investigation of a case of eczema. The treatment is to cut down the amount of fat in the diet. At times, the evidences of carbohydrate indigestion are found, in which case the percentage of sugar must be reduced.

In children having eczema toward the end of the first year, or during the second year, three principal dietary errors are found, namely, general overfeeding, too much solid food, or too much starch-containing food. In the first case the diet should be reduced to correspond with that appropriate to the child's age, and if symptoms of indigestion are then still present, the diet should be still further reduced. If too much solid food is being given, its amount must be reduced, and the quantity of milk in the diet must be increased. When the cause is too much farinaceous food, evidence of the correctness of the diagnosis is often afforded by the microchemical examination of the stools, which shows the presence of excessive starch. In such a case, farinaceous food must be greatly restricted, the diet consisting of milk, beef juice or broth, eggs, a little meat, fruit, and bread or zwieback in moderate amounts.

The condition of the bowels requires careful attention in the treatment of eczema. At times constipation is the principal exciting cause, the removal of which is the only measure necessary to bring about a cure. Such cases should be treated as described under the heading of constipation. In babies under one year, I prefer milk of magnesia, given in divided doses with every feeding. In older children my preference is for sodium phosphate, given in a single dose each morning. The occasional bringing about of catharsis by the giving of calomel is useful, particularly in cases of overfeeding.

Whenever the urine is scanty and concentrated, or highly acid, it is well to see that the child takes plenty of water between feedings. Also, ten to twenty grains of acetate of potassium should be given daily.

The general treatment of the causes of eczema operating from within, may be summed up in these words. The general condition

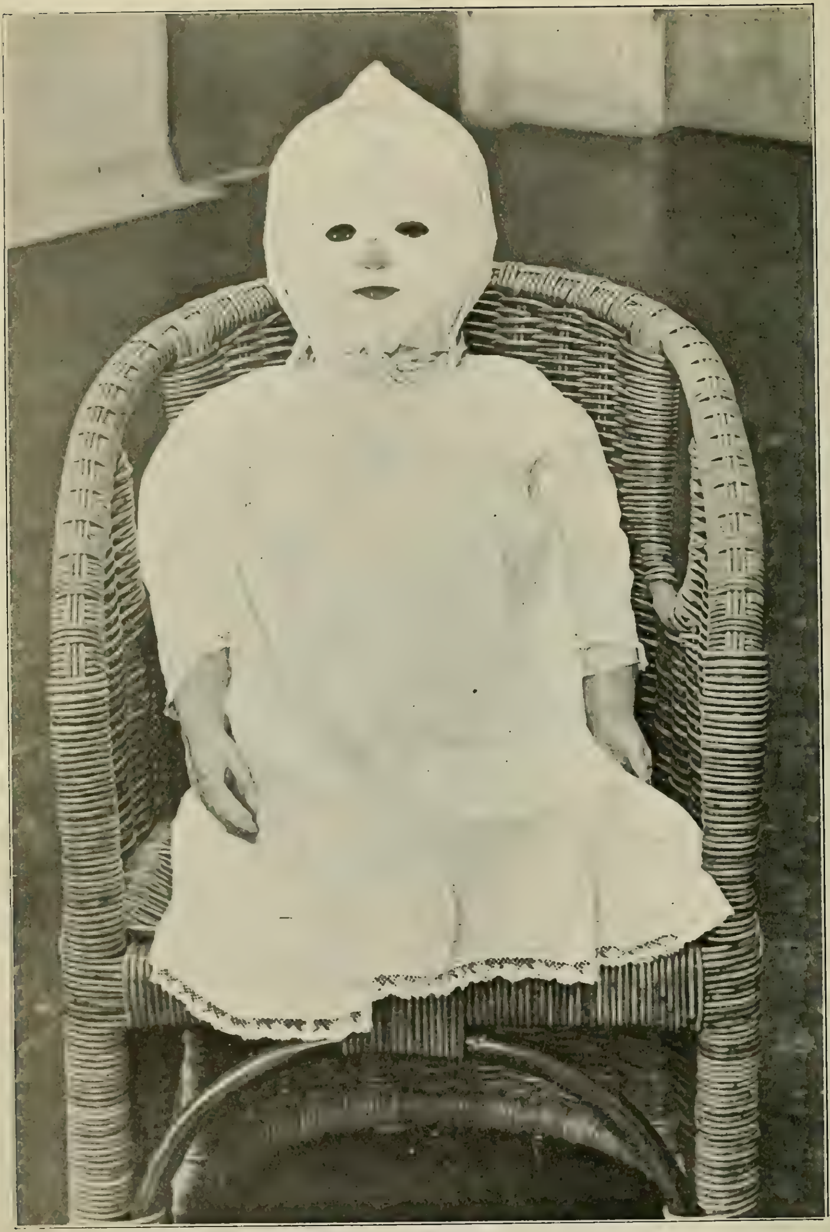


FIG. 268—Mask and hood for the treatment of eczema

of the child should be made as good as possible. The form of eczema most likely to be benefited by dietary treatment is the common facial exudative eczema. Towle and Talbot have recently obtained evidence that dietary measures are less affective in the subacute or chronic papular form.

LOCAL TREATMENT.—The principal object of the local treatment of eczema is to protect the skin from external irritation. In a case of eczema, the skin is so sensitive to external irritants, that such conditions as contact with water, or even normal exposure to the air, may cause irritation of the skin. A case of eczema should never be washed with plain water. The good results in eczema which follow the use of particularly medicated ointments or pastes are usually attributed to the composition of the remedy. I believe that they should be rather attributed to the protection of the skin from even such slight irritation as contact with the air. The chief variety of external irritation in eczema which produces the disease, and which most retards recovery, is rubbing and scratching. Therefore our first object must be to prevent all mechanical irritation.

A mask is the most useful means of preventing both scratching, and exposure to the air. It is made of linen, in which are cut holes for the eyes, nose, and mouth Under it may be applied whatever ointment or paste is used, and it is kept in place by a cap. (Fig. 269). It is also necessary to prevent the child from getting the hands to the face. When there is a tendency to scratch, mittens are not enough, but the hands must be mechanically restrained. This can be accomplished by applying to the elbows pasteboard splints held in place by bandages. A still better method, is to have the sleeves of the child's night-dress long enough to come below the hands, and closed at the ends. The ends of the sleeves are fitted with tapes, which when tied to the sides of the crib, permit movement of the hand and arm within the sleeve, without permitting the hands to reach the face. This method has the further merit of preventing the child from turning over and burrowing with the face in the pillow. The mask and mechanical restraint sometimes seem cruel to the mother when the child is very uncomfortable from itching. The itching is, however, largely kept up by the rubbing and scratching, and when this is prevented, the child is soon manifestly much relieved. Many of the local applications advocated in eczema are supposed to be antipruritic, but I believe their apparent results are largely due to prevention of scratching.

The first step in the treatment of a case of infantile eczema of the face and scalp, is the softening and removing of the scales and crusts. This can usually be accomplished by applying olive oil for twelve to twenty-four hours, and then removing the crusts by washing with equal parts of water and lime water.

At the very beginning of an acute vesicular eczema, dusting powders may be used. Various combinations of talcum powder, starch, boric acid, zinc oxide, and bismuth subnitrate may be employed. A useful preparation is—

℞

Talci Venet.	
Zinci Oxidi	aa ʒ iv
Amyli	℥ i

Soothing lotions are recommended for the very acute stages. Such a preparation is—

℞

Zinci Oxidi	℥ ss
Glycerini	ʒ i
Acidi Carboloci	ʒ ss
Aquae Calcis—q.s. ad	℥ viii

This may be sopped on every hour and left to dry on. If there is not much itching, the carbolic acid may be left out.

Black wash is a very soothing application in the acute stage of these infantile eczemas. My routine treatment in these cases, is to use a simple protective ointment applied on a mask. During the acute inflammatory stage, the mask is removed four times a day, and black wash, either in full strength, or diluted one-half with lime water, is sopped on. Then the mask, with fresh ointment, is reapplied. The protective ointment which I prefer is the following:

℞

Bismuth Subnitrate	
Aquae Calcis	
Agnine	
Vaseline	aa ℥ i

After the most acute stage has subsided, more stimulating applications are indicated. Of the great variety of stimulating agents recommended I prefer tar in the resistant facial eczemas of infants, and crude tar appears to work better than any refined preparation. The following ointment may be tried:

℞

Crude coal tar	ʒ ss to ʒ ii
Zinci Oxidi	ʒ i
Amyli	ʒ ii
Vaseline	℥ i

The quantity of tar should be very small at first, and, if the process shows no tendency to become more acute, should be gradually increased.

When cases of facial eczema recover, the greatest care must be taken to prevent a recurrence. Even if the skin has become perfectly clear, the dietary measures must be continued, and the skin must be guarded as carefully as possible from external irritation. One scratch will set a case of eczema back at least a month. I begin to leave the mask off little by little, letting the child go without mask or ointment each day, for a short time, which is gradually increased. Even then, the face should be protected with ointment when the child goes out doors, and exposure to cold winds must be avoided for a long time.

The subacute and chronic papular eczemas of the body and limbs may be treated in much the same way. Scratching must be prevented. During the acuter stages, the bismuth ointment given above may be used. When the acute stage has subsided, itching is often very severe. In this stage the following paste may be used:

℞

Acidi carbolici	gr. x
Hydrarg. chloridi mitis	gr. xv
Amyli	
Zinci oxidi	aa ʒ i
Vaselini	℥ i

When the process becomes more chronic, the crude coal tar ointment should be used as in facial eczema.

For pustular eczema of the scalp, a white precipitate ointment is the best application.

℞

Hydrarg. Ammoniat	gr. v to xx
Vaselini	℥ i

TREATMENT OF INTERTRIGO.—The most important point in the treatment of this condition is the most scrupulous care in the changing of napkins as soon as they become soiled. In mild cases, still in the stage of erythema, the skin must be kept dry and clean. Soap and water should not be used on the parts affected, but they should be washed with water and lime water, equal parts, and dusted with an absorbent powder, such as—

℞

Acidi borici	ʒ i
Zinci stearat	ʒ ii
Talci venet	℥ i

If this treatment is not effective, and the skin is more acutely inflamed, a wash may be applied and left to dry on.

℞

Zinci oxidi	
Calamniae preparat	aa ʒ ii
Aquae calcis	℥ viii

If the process has become more severe, so that eczema has developed, a zinc oxide ointment may be applied on a cloth. Sometimes, in these severe cases, a single application of a 2 per cent silver nitrate solution may be tried, and then the other measures are continued. In very acutely inflamed cases, in which the condition of the skin resembles that seen after a superficial burn, carron oil may be applied, till the very acute stage has subsided. Olive oil or castor oil may be used instead of carron oil.

PROBLEMS AND RESEARCH.—The obscure factors in the etiology of eczema do not at present offer a very encouraging outlook for research. The particular problems are the following. 1 Is there a congenital constitutional peculiarity predisposing toward eczema, and if so, what is its nature? 2. What is the nature of the products of disturbed metabolism which appear to act as predisposing or exciting causes of eczema? Both of these problems are connected with the most difficult and obscure region in the whole field of medical research. For light on the subject, we can only look to the sort of researches on metabolism which are being carried on in some of our nutrition laboratories—researches such for example as those which have been throwing light on diabetes mellitus.

There is a field at present for clinical investigations on the relation of eczema to diet and to certain definite forms of disturbance of digestion. If, for instance, it can ever be shown positively that certain cutaneous manifestations of eczema are associated with dietary errors, or particular disturbances of digestion, advance will have been made.

DERMATITIS

Dermatitis is an inflammatory affection of the skin, produced by some recognized cause. The lesions are, as a rule, fugitive, and with a few exceptions are not characterized by an especial and peculiar form of efflorescence. The course of the disease, and the recognition of some definite exciting cause, enables us to distinguish this condition from others which resemble it.

Clinically the group may be divided into a *dermatitis traumatica, dermatitis venenata, dermatitis calorica,* and *dermatitis medicamentosa.*

DERMATITIS TRAUMATICA.—Dermatitis traumatica is the term applied to the local reaction which takes place in the skin, following some trauma, either slight or severe, such as may result from pressure, friction, or direct blows. The lesions vary from a simple, temporary erythema to deep and extensive ulcers, as in certain bed-sores.

DERMATITIS VENENATA.—Dermatitis venenata is the name applied to those dermal lesions which are caused by the external application or contact of irritating substances. The inflammatory condition may be due to simple *mechanical* irritation, such as is sometimes caused by the lodgement in the skin of small particles of matter; or it may be due to a *poison*, arising either from emanation of a poisonous volatile principle or from actual contact. *Ivy poisoning* is one of the most common and important examples of this class of affections. It is caused by contact with the leaves of the *rhus toxicodendron.* The cases are most common in the autumn, probably owing to the fact that people are tempted to gather the leaves because of their brilliant coloring at this season of the year. There is some difference of opinion as to the volatility of the poisonous active principle. *Rhus venenata,* or poisonous oak, is another common cause of plant poisoning.

Some of the more important irritants which may produce a dermatitis venenata are *chrysarobin*, an effective remedy in psoriasis; also preparations of carbolic acid, turpentine, iodine, mercury, and sinapis. There are sixty or more drugs (White) cited as more or less frequent causes of a dermatitis. Knowing the great delicacy of an infant's skin, we cannot be too cautious in prescribing ointments and applications, and should always seek for the cause of a dermatitis in some article which is of common use perhaps, but at times an undoubted source of irritation to the skin.

SYMPTOMS OF IVY POISONING.—The eruption appears generally within a day or two after exposure, with redness, edema and papules, which pass rapidly to the stage of vesicles, which may become pustular from a secondary infection. The course of the disease is from two to six weeks. The face, hands, and genitals are especially liable to be the seat of the disease, the extension to other parts of the body taking place by means of the hands. Itching is intense.

DIAGNOSIS.—The lesions of ivy poisoning resemble those of an acute eczema. In making the diagnosis especial attention should be paid to a history of exposure to ivy, the time of year, the asymmetry in the distributions of the lesions, the severe itching, and the history of previous attacks. An especial susceptibility will often aid in the exclusion of other similar conditions, especially eczema.

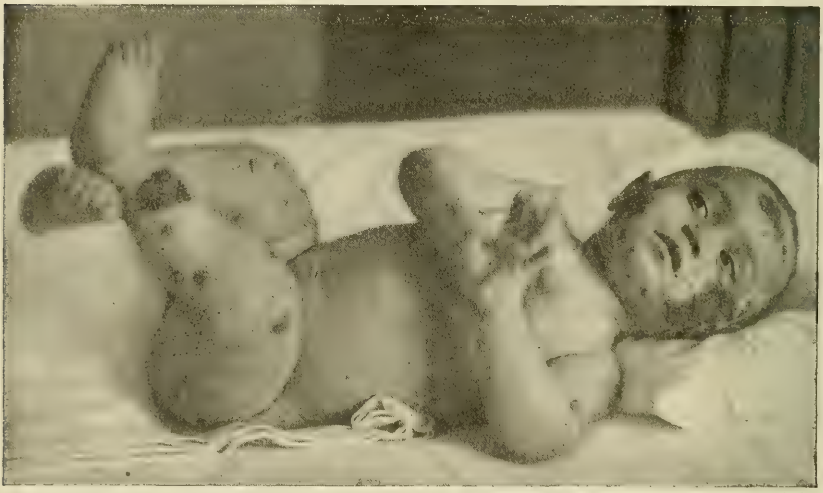
TREATMENT.—There is no specific remedy. The treatment is the same as in an acute eczema, except that it is well to begin by washing the skin thoroughly with soap and water.

DERMATITIS CALORICA.—Dermatitis calorica is a form of dermal inflammation produced by the milder degrees of heat. The action of the sun's rays is the simplest and most common type met with in children. The lesions in sunburn may vary from a simple erythema to vesicles and bullae, depending upon the intensity of the heat, the duration of exposure, and the susceptibility of the skin in an individual case. Diminution of heat may produce lesions the character and course of which are very similar to those which result from an excess of heat. Chilblains are a very common example o the result of a diminution of heat. They occur chiefly in children with feeble circulation who wet or chill their feet, and then suddenly heat them. In such instances the skin is red, soft, and boggy. The boggy areas may break down and form indolent ulcers, which are difficult to cure.

TREATMENT.—The treatment of these cases of dermatitis calorica is the application of some simple lotion, or ointment.

DERMATITIS MEDICAMENTOSA. Dermatitis medicamentosa is a general term including a great variety of lesions produced by the action of certain drugs administered internally. Arsenic may produce an efflorescence of a very complex character which may be erythematous, papular, urticarial, bullous, pustular, and even hemorrhagic. It may also give rise to herpes zoster, as a result of certain changes in the nerve-endings. *Belladonna* often produces an efflorescence of an erythematous character resembling closely that which occurs in scarlet fever. The *iodides* are especially likely to cause an efflorescence of *acne*. The bromides often cause lesions of a papular or pustular character. Both the bromides and iodides occasionally cause lesions of a most unusual character. *Chloral*, *digitalis*, *opium*, *quinine*, the *salicylates*, and many of the recent new remedies sometimes cause an efflorescence. The erythematous, urticarial, and papular efflorescences which are seen at times after the administration of *antitoxin* will be more fully described under serum rashes.

FIG. 270



Dermatitis Medicamentosa in an infant of eleven months, from the supposed administration of fifty grains of bromide of potassium in the course of three days

TREATMENT.—The treatment consists in discovering and discontinuing the drug the use of which is causing the irritation of the skin.

III. THE TOXIC SKIN DISEASES

There is a group of skin diseases, the manifestations of which are of such a character, as to suggest that they are caused by some toxin circulating in the blood. In the case of the serum eruptions, a toxic etiology is definitely established. Several other diseases in this group so closely resemble the serum eruptions in their clinical manifestations, that we are justified in assigning to them a similar etiology, although the origin and nature of the toxin is more obscure. There is some further evidence of the toxic nature of these conditions to be drawn from their occasional association with recognizable errors in diet, or disturbance of metabolism.

The diseases in this group which are seen in infancy and childhood with sufficient frequency to warrant their description here, are the following: Serum eruptions, Urticaria, Erythema Multiforme, Erythema Nodosum, Angioneurotic Edema, and Herpes Zoster.

THE SERUM ERUPTIONS

ETIOLOGY.—These eruptions are due to the injection of the serum derived from an animal of another species. The rash is only a part of certain general toxic manifestations. The full description of the phenomena of "serum sickness," and the consideration of the relation of these phenomena to the subject of anaphylaxis, have been given in the article on Diphtheria. We have here to consider only the cutaneous manifestations.

SYMPTOMS.—The rash may appear at the earliest in one day after the injection of the serum, or it may be delayed as long as three weeks. The larger number of rashes appear from the sixth to the twelfth day. The appearance of the rash is usually accompanied by constitutional symptoms, such as elevation of temperature, headache, pain in the joints, and a certain amount of prostration. The symptoms last usually from twenty-four to seventy-two hours.

The majority of rashes are of an urticarial character, and either show large typical wheals, or patches of urticarial erythema. Next in frequency occur rashes showing a polymorphous erythema, with non-elevated patches of redness having sharply defined irregular margins. There may be a tendency to circular formation, with pale centers. The resemblance to erythema multiforme is often close. In some cases rashes are seen which bear a close resemblance to the exanthemata of measles and scarlet fever. Vesicular and bullous lesions occur, but are rare, and purpuric eruptions are very rare. Papules and vesicles are sometimes seen. Edema of the skin is fairly common, involving most commonly the face, but sometimes seen in the penis, scrotum, hands, and feet. Most of the serum rashes are accompanied by very *severe itching*.

The cutaneous manifestations of serum sickness resemble the eruptions seen in three other supposedly toxic diseases of the skin, namely, Urticaria, Erythema Multiforme, and Angioneurotic Edema. All the varieties of eruption seen in these three diseases may appear as serum rashes.

The distribution of the eruption is so extremely irregular, that it needs no detailed description. Any part of the body may be involved or spared.

PROGNOSIS.—The rash disappears rapidly, with subsidence of the constitutional symptoms. The duration of the rash is variable. Two days is an average duration, though it may persist somewhat longer.

TREATMENT.—In young children, no treatment is required. In older children, the itching is more annoying, and may require measures for relief. A warm bath with water containing a little washing soda, is often of great service. The following lotion may be used, if itching is very severe:

℞

Acidi Carbolici	ʒ ss
Zinci Oxidi	ʒ ii
Glycerini	ʒ i
Aquae Calcis	℥ viii

URTICARIA

(Hives). (Nettle Rash).

Urticaria is a disease characterized primarily by the formation of evanescent edematous elevations of the skin, and by severe itching.

ETIOLOGY.—Urticaria is a vaso-motor neurosis, the lesions being due to excitation of the vaso-motor system. There is no reason to doubt that the immediately exciting cause is a toxin, brought to the vaso-motor apparatus by the circulating blood. The toxin is in most instances derived from the alimentary tract.

Certain particular articles of diet may be the source of the toxin, acting as poisons in certain persons. Individual susceptibility plays an important part. Certain individuals can never eat particular articles of diet without an attack of urticaria. Urticaria may similarly be caused in certain persons by the taking of a particular drug; this cause is, however, comparatively rare in early life Those articles of diet most frequently associated with urticaria in children are acid fruits, strawberries, oatmeal, and shell-fish.

Urticaria in early life is frequently associated with gastro-intestinal disturbances. In these cases the toxic nature of the exciting cause is probable, but not proven. The disease is most common in indigestion due to overfeeding with carbohydrate, but may be seen in other forms.

Urticaria is associated with conditions in which a reflex rather than a toxic excitation of the vaso-motor system is suggested. In gastro-intestinal disorders, it has been assumed that mechanical irritation of the stomach and bowels may be the exciting cause of urticaria. In these particular conditions, however, the rôle of reflex excitation is rendered doubtful on account of the impossibility of excluding toxic absorption. In cases of urticaria associated with intestinal parasites, and with sources of reflex irritation in other parts of the body than the alimentary tract, reflex excitation of the vaso-motor system is a possible etiologic factor, although not supported by as great a weight of evidence as is toxic excitation.

Urticaria may be produced by excitation coming from without, as in the sting of a nettle, contact with a jelly-fish, and the bites of wasps and mosquitoes. In these cases, the action of the excitant is probably toxic rather than reflex. I have seen a general severe urticaria follow the sting of a bee.

The susceptibility of certain individuals to certain toxins, and the fact that urticaria tends to be recurrent in certain individuals, suggest that there is an underlying constitutional predisposition to urticaria, which plays an important etiologic rôle. Such a predisposition, if it exists, consists in an abnormal excitability of the vasomotor apparatus.

SYMPTOMS.—The manifestations of urticaria in children present certain features which are quite different from those seen in adults. The difference is particularly marked in infancy. The typical initial lesion of urticaria is the wheal, a firm, circumscribed whitish or pinkish elevation, with a reddish areola, which appears suddenly, and persists from a few minutes to several hours, and then disappears. In children, owing to the susceptibility of the skin, the duration of the wheal as such is very brief, and in many cases no typical wheals are seen at any time. The lesion most frequently found in children is a hard skin-tinted or pinkish papule, with a reddish areola. The areola soon disappears, but the papule persists. The papules may be crowned with a vesicle. In any case, the summits of the papules are apt to become excoriated by scratching.

The distribution of the eruption is very variable. It may be general, or confined to certain definite parts of the body.

The itching is very intense. The constant scratching leads to secondary irritation of the skin. so that an accompanying dermatitis is often seen, or, especially in poorly nourished children, pustular or ulcerative lesions. In some cases, through secondary infection, the lesions closely resemble those of impetigo contagiosa, with which they are in fact identical, the only difference being the original urticarial origin.

As the initial lesions are common on the hands and feet, the character of the eruption and its distribution as seen in children, is often strongly suggestive of scabies.

In infants especially, the disease may become more or less chronic, a few fresh lesions appearing from time to time for a long period. In such cases the primary character of the lesion may be greatly masked by the secondary lesions produced by scratching.

DIAGNOSIS —Cases of acute generalized urticaria beginning with recognizable wheals, present no difficulty in diagnosis. Even cases which have become chronic, and show a variety of secondary lesions, may be easily diagnosed when the fresh lesions appearing from time to time are recognizable as wheals. When, as is common in such cases, the initial lesion is a papule, or vesicle-crowned papule, the diagnosis may be quite difficult. The main thing is to remember that in children, more or less generalized, itching eruptions are due most commonly to eczema, scabies, or urticaria. The lesions do not resemble eczema.

Scabies is either recognized by finding the burrows, or excluded by finding initial wheals or papules sufficiently characteristic of urticaria.

Varicella may be suggested by a vesicular urticaria, but in varicella there is no such intense itching, and characteristic initial vesicles may usually be found.

PROGNOSIS.—In an acute outbreak the prognosis is good, and the physician may confidently predict that the attack will be over in a few days. Chronic cases are very persistent, fresh crops of lesions tending to appear in spite of the most careful treatment, and the disease may continue resistant for years.

TREATMENT.—The treatment of urticaria is local and general. The local treatment is in no way curative, but is simply designed to relieve the itching, and prevent the formation of secondary lesions by scratching. The general treatment is designed to cure chronic cases, or to prevent recurrence.

LOCAL TREATMENT.—For the relief of the itching a great variety of applications have been recommended, containing such antipruritics as carbolic acid, alcohol, menthol, subacetate of lead, and so forth. A good routine in practice is to order a tablespoonful of washing soda to be added to the water of the child's bath. The following lotion is as good as any for routine use:

℞

Acidi Carbolici	ʒ ss
Zinci Oxidi	℥ ss
Glycerini	ʒ ii
Aquae Calcis	℥ viii

This is applied and allowed to dry on the skin.

GENERAL TREATMENT.—Acute attacks require no special treatment. It is well to give at the outset a dose of castor oil or some saline cathartic, and to put the children on a milk diet until the attack has subsided.

The persistent chronic and recurrent forms of urticaria seen in children are very intractable. The first step is the treatment, by dietary measures, of any form of indigestion which may be present. In infants, the microchemical examination of the stools for fat and starch may give valuable suggestions as to the dietary changes which should be made. In older children the diet must be closely scrutinized, and any article of diet which is suspicious, either on account of its known occasional association with urticaria, or because of known occasional association with indigestion, should be excluded.

The bowels should be kept freely open. Magnesia in infants, and sodium phosphate, or some stronger saline, in older children, should be given each morning. An occasional dose at night of castor oil or calomel is useful.

If the urine is highly acid and scanty, potassium acetate should be given.

All causes of local or reflex irritation should be removed. The sleep may be so much disturbed as to require trional or bromide. In some cases, usually in children from one to six years of age, all the ordinary dietary and hygienic measures fail to relieve urticaria, and it remains persistent in spite of the correction of all the ordinarily probable causes. In such a case the child must be put on an exclusive milk diet. On this the disease is usually relieved. The other articles of food are then added one by one, until the recurrence of the urticaria betrays the particular article of diet which cannot be tolerated by the individual child.

ERYTHEMA MULTIFORME

This is an acute inflammatory disease of the skin, characterized by a variety of lesions, of which one type predominates in a particular case.

ETIOLOGY.—All the features of the disease point toward a toxin circulating in the blood as the cause. The nature of the toxin and its mode of action are unknown. The disease has been associated with certain drugs, with poisoning from ptomains in food stuffs, and disturbances of metabolism in various diseases of the viscera. Except as a manifestation of serum sickness, it is rarely if ever seen in infancy, occurring most often in the later years of childhood.

SYMPTOMS.—The attack may be preceded or accompanied by mild constitutional disturbances, such as slight fever, malaise, or arthritic pain. The eruption is very variable and may consist of macules, papules, vesicles, blebs, or hemorrhages. One type of lesion only is usually seen in each case. Any part of the body may be involved, although the extensor surfaces of the hands, feet, arms, and legs, are particularly liable to be affected. In children, eruption about the buttocks is fairly common. The eruption runs a course of from one to four weeks. The disease as it occurs in children presents no essential difference from adults.

DIAGNOSIS.—The diagnostic features of the disease are the tendency of the lesions to assume a *bluish-red* or *purplish color*, and the tendency toward *an annular configuration with lighter colored, or clear, centres.* It is further distinguished from urticaria by the greater persistence of the lesions, the predilection for certain regions, the absence of distinct wheals, and the absence, or very slight grade, of itching.

PROGNOSIS.—The prognosis in children is always favorable. Periodic recurrences are sometimes seen.

TREATMENT.—Itching is usually not severe enough to require treatment. If it is marked, the treatment is the same as in urticaria. The general treatment should be that of an acute attack of urticaria, keeping the bowels freely open, and the kidneys acting properly.

PROBLEMS AND RESEARCH.—There is undoubtedly a very close family relationship between erythema multiforme, urticaria, angioneurotic edema, and possibly purpura. Osler has published several communications on visceral manifestations associated with erythema multiforme, and with angioneurotic edema. Such observations raise into importance the question whether there may not be certain internal disorders responsible for this whole group of diseases, which may manifest themselves at one time by erythema multiforme, and in subsequent attacks by urticaria, angioneurotic edema, or purpura.

ERYTHEMA NODOSUM

This is an acute inflammatory disease of the skin, in which the characteristic lesion is a hard, tender, node-like swelling. Some writers classify it as a variety of erythema multiforme.

ETIOLOGY.—The disease has been associated with arthritis, with gastro-intestinal disorders, and with general disturbances of nutrition. It has been especially considered a "rheumatic" manifestation, but as our present views regard the vague term "rheumatic" as applying to a great variety of arthritic and muscular affections, most of which are now regarded as being of toxin origin, the term "rheumatic" as applied to erythema nodosum is equivalent to toxic. The nature and source of the toxin remain unknown. It is probable that like erythema multiforme, this disease also may be produced by a great variety of poisons.

The disease is not seen in infancy or early childhood, usually occurring in the later years before adult life is reached. Females are affected more frequently than males.

SYMPTOMS.—The disease is usually ushered in by mild constitutional symptoms. There may be moderate fever, malaise, and articular pains. The lesions are most frequently seen in the tibial regions, though occasionally they are found on the forearms, trunk, or face. The nodes are round or oval, rose-red in color, tense and shining, and *very tender to the touch.* The induration appears to be notably subcutaneous, or at least involving the deeper layers of the skin The lesions last a week or ten days, during which time they gradually soften, and undergo *all the color changes seen in a common bruise*

DIAGNOSIS.—The disease is distinguished from bruises, abscesses, and gummata, by the peculiarities of distribution, tenderness, and color changes.

TREATMENT.—The bowels should be kept freely open, and sodium salicylate should be given internally. The limbs should be kept elevated. Applications do no good.

ANGIONEUROTIC EDEMA

(Acute Circumscribed Edema) (Localized Toxic Edema)

This is a condition characterized by the rapid appearance of circumscribed edematous swelling.

ETIOLOGY.—This disease is of toxic origin, and is so closely allied to urticaria, that it is a question whether it is not a particular manifestation of that disease. Toxic edemas are not uncommon in early life. In infancy especially, associated with severe disturbances of nutrition, are seen generalized edemas of undoubted toxic origin, resembling the edema seen in nephritis. Angioneurotic edema is distinguished from these by its localized character, and is rare in early life.

SYMPTOMS.—The swellings come on suddenly and rapidly The face is most commonly involved, but large areas in other parts of the body, or even the whole of a limb, may be affected. The mucous membrane of the respiratory tract may be affected in rare cases, causing suffocative symptoms. The edema may involve the alimentary tract, causing gastroenteric symptoms Itching is not marked The lesions disappear after several hours or days.

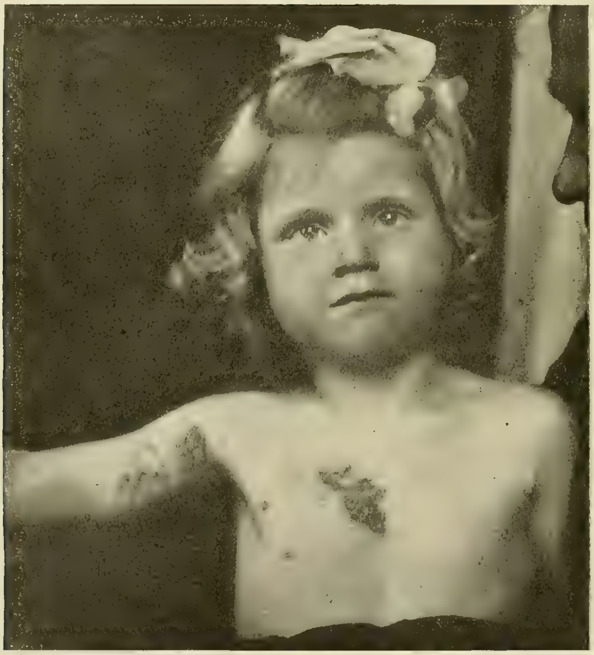
TREATMENT.—The treatment is that of urticaria.

HERPES ZOSTER

(Shingles)

This is an acute inflammatory disease, characterized by the formation of groups of vesicles over the areas of distribution of cutaneous nerves, and by neuralgic pain.

FIG. 271



Showing the lesions of herpes zoster in a girl of 4 years

ETIOLOGY.—The disease is due to a lesion involving sensory nerve structure. There may be a simple inflammation of peripheral nerve structure, or of the sensory ganglia of the posterior spinal nerve roots, with a descending interstitial neuritis. It has been associated with exposure to cold, with mechanical injury to nerve structure, with the continued use of arsenic, and with certain infections. The cause is therefore, either traumatic or toxic. When toxic, the source of the toxin is probably most often various infections. Toxins of non-infectious origin cannot be excluded from a possible etiologic rôle.

SYMPTOMS.—There is often more or less severe prodromal pain, which in children usually ceases with the appearance of the eruption, though it may persist during the period of efflorescence. In children pain is often not complained of at any time.

The eruption consists of irregular groups of vesicles of a size varying from a pin-head to that of a pea. The vesicles appear in crops and follow in an interrupted manner the course of distribution of the sensory nerves. The vesicles rest on an inflamed and reddened base. They do not tend to spontaneous rupture, but after a few days dry, leaving yellowish-brown crusts, which fall off, usually leaving no permanent scar.

The eruption is *unilateral* Any part of the cutaneous surface may be affected, although the disease is most frequently seen in the region supplied by the intercostal, lumbar, and trifacial nerves.

The neighboring lymphnodes are usually enlarged. There may be slight fever, and loss of appetite.

DIAGNOSIS.—The clinical features of the disease are so characteristic, that the diagnosis is easier than in any other skin disease.

PROGNOSIS.—Favorable. Most cases recover within two weeks, though some may persist as long as three weeks.

TREATMENT.—Diet and hygiene should be regulated as in any disease of possible toxic origin. Local treatment is directed toward protecting the inflamed areas from injury and infection. They may be kept covered with an ordinary dusting powder, and protected with absorbent cotton held in place by a bandage. Or the area may be painted, if not too extensive, with collodion containing ichthyol in proportions of one drachm to the ounce.

IV. THE PARASITIC SKIN DISEASES

There are a number of diseases of the skin occurring in early life as well as in adult life, of which parasites are definitely known to be the cause. The parasites may be vegetable, (bacteria or fungi), or animal. The diseases due to the vegetable parasites are Furunculosis, Impetigo Contagiosa, Gangrenous Dermatitis, and Tinea. Those due to animal parasites are Scabies and Pediculosis. There is another group in which a parasitic origin is considered probable, although it is not proven. The diseases of this group seen in children are Seborrheic Eczema and Verrucae. Finally there is a group of cutaneous affections in which a parasitic etiology has been suggested as possible, although unsupported by any important evidence. The etiology of these conditions is really wholly unknown. Of this group, the only disease occurring often enough in children to be considered here is Seborrhea

FURUNCULOSIS

ETIOLOGY.—A furuncle or boil, is an acute circumscribed inflammation in the deeper layers of the skin which originates about a hair follicle, or about a sweat or sebaceous gland. It is caused by infection with some pyogenic microörganism, usually the staphylococcus aureus. As staphylococci are present upon the normal skin, there must be some other factor in the occurrence of infection. This other factor is a lowering of the resisting power of the skin, which may be either local or part of a general lowering of resisting power.

Such lowering of resisting power occurs with especial frequency in young children, more particularly in infants, and manifests itself by the tendency toward the formation of large numbers of small furuncles. It is to this condition that the term *furunculosis* is applied.

The delicacy of structure of the skin in infancy must also be invoked in explaining the frequency of furunculosis as the particular manifestation of lowered resistance to infection.

The condition may occur in children who are apparently healthy in all other respects. It is, however, much more frequently seen in poorly nourished or debilitated infants.

SYMPTOMS.—The furuncles appear in successive crops. They may appear upon any part of the body, but are seen most frequently and in greatest numbers on the scalp, face, and shoulders. Their number may run into the hundreds. Their size varies from that of a pea to that of a walnut, and they differ from the boils seen in later life in that they do not usually contain a core.

PROGNOSIS.—The disease may last during a period of months. It is in general not serious, but in infants suffering from severe malnutrition, or having very low resisting power, the process may become a gangrenous dermatitis, which is much more serious.

TREATMENT. LOCAL TREATMENT.—The parts affected should be frequently bathed with soap and water followed by a 1 to 5,000 solution of corrosive sublimate. I prefer to keep them covered with boric acid powder, and not to use any wet dressing. The best method of treating the multiple small furuncles is to wait until each one points, and then to empty it, but in my experience, the pustules heal much more rapidly if they are not opened by incision, but by pressure. When the white spot crowning the boil has appeared, the two thumbs are applied on each side, and the furuncle pressed flat. The compression should be hard enough to cause all the contents of the boil to spurt out, and should be maintained for several minutes. Only large abscesses which do not point in a definite small area need to be incised. After being emptied by pressure, the boils should be covered freely with boric acid powder.

GENERAL TREATMENT.—The internal administration of sulphide of calcium has been widely recommended as having a direct action on the disease, but has given no final evidence of having any value. Holt believes that the internal administration of yeast has given evidence of striking benefit, but in a disease of this kind tending toward eventual spontaneous recovery, such observations must be repeated many times to be convincing. My personal limited experience with the use of yeast gave no evidence of therapeutic value.

General treatment at present must consist in the correction of all faulty conditions in the hygiene of the infant, and in the most careful attention to its nutrition. Iron should be given if there is anemia.

VACCINE TREATMENT.—The most convincing evidence of therapeutic benefit derived from vaccine therapy is found in staphylococcus infections. I believe this to be the best method of treating furuncles, and that it should be employed in all prolonged or resistant cases. Stock staphylococcus vaccines have repeatedly appeared to have been followed by improvement, but an autogenous vaccine is always preferable. The dose in furunculosis should be 50,000,000 cocci, and this should be repeated in about six days. Improvement, if it occurs at all, should be evident after the second injection. Evidence of improvement is seen in the more rapid healing of the emptied furuncles, and in the non-appearance of new ones. Further injections need not be given unless the disease shows a tendency to grow worse.

IMPETIGO CONTAGIOSA

This is an acute infectious and contagious disease of the skin, characterized by the formation of superficial vesicles, which quickly become pustular, and dry as yellow crusts.

ETIOLOGY.—There is difference of opinion as to the microörganism causing impetigo contagiosa. Staphylococci have been found by some observers; streptococci have been found by others, while still others have found both staphylococci and streptococci associated with the lesions. One theory holds that the disease represents a particular reaction of the skin to more than one microörganism, the definite character of the lesion being due to the fact that only the most superficial layers of the skin are infected. Another theory holds that the disease is due to a specific microörganism. In favor of the latter theory are the facts that impetigo contagiosa is a distinctly contagious disease characterized by definite lesions; that it can be transmitted by inoculation from one individual to another, and by autoinoculation from one part of the body to another; finally that when so transmitted, the same definite lesion is reproduced. Among those who believe in the specific character of the microörganism causing the disease, there is further difference of opinion, some believing that the staphylococci and streptococci associated with the disease are not the cause, but that the cause is wholly unknown, while others believe that one of the organisms found associated with the disease is the cause, and represents a specific strain of that örganism. In favor of the former view is the fact that none of the other reactions usually seen with staphylococcus or streptococcus infection is commonly observed in impetigo contagiosa. In favor of the latter view is the fact that streptococci at least are divisible into numerous strains, some of which are known to produce specific tissue reactions.

The disease is most common in children, particularly in children of the poorer classes. It is readily transmissible by accidental inoculation It tends to occur in groups, affecting several children in one family, or in one building. I do not believe, however, that the disease is contracted solely by inoculation from another case; some cases give every evidence of being contracted by autoinoculation from the discharge of a coryza.

SYMPTOMS.—The lesions first seen are vesicles or blebs, which rapidly, usually in a few hours, become pustular. The vesicles are flat, and appear flaccid and wrinkled; in size they vary from a pinhead to a bean. There is usually no areola. The covering of the vesicles is very thin, and they rupture very easily, drying and forming yellow, superficial crusts, which have the appearance of being "stuck on" the surrounding healthy skin. The crusts finally become detached, and fall off, leaving a reddish spot which disappears in a few days. The lesions are scattered at first, but in places often coalesce so as to form fairly large crusted areas. The lesions are seen most frequently on the face, neck, and hands. Occasionally they are seen on the feet, and trunk. New lesions keep appearing through autoinoculation. In infants quite large blebs are sometimes seen, and these often rupture without forming crusts, leaving only spots where the most superficial layer of the skin has been lost. Many cases in infants diagnosed as pemphigus are really this form of impetigo.

DIAGNOSIS.—The disease is recognized by the superficial character of the lesions, the discreteness and flaccid character of the vesicles, the characteristic situation on the face and hands, and the absence of marked itching. *Pustular eczema* is itchy, does not yield so readily to treatment, and the lesions have a reddened base. *Varicella* has smaller vesicles with reddish areolae, and has a more general distribution; it begins usually with fever, and lesions are usually found in the mouth. *Pemphigus* is a serious chronic disease, rare in children, with large distended blebs.

PROGNOSIS.—The disease yields very easily to treatment.

TREATMENT.—The crusts are removed with soap and water; if they are very thick they should first be softened over night with vaseline. The following ointment is effective:

℞

Hydrag. Ammoniat..................	gr. v to gr. xx
Vaseline...........................	℥ i

The child should be prevented from picking at the lesions.

DERMATITIS GANGRENOSA INFANTUM

(Varicella Gangrenosa). (Multiple Disseminated Gangrene of the Skin in Infants)

This disease may be secondary to any of the pustular diseases of the skin in infants. It is most frequently seen after varicella. The lesions are crusted pustules with inflammatory areolae. When the crusts are thrown off, loss of tissue appears, and the ulcers leave permanent scars. The lesions are seen most often on the neck and chest. This disease occurs only in infants of very poor vitality, and runs a course of several weeks. The constitutional symptoms of septic infection may be present. Many cases end fatally.

TREATMENT—Every possible dietary and hygienic regulation which can aid in building up the infant's health should be employed.

The lesions should be kept clean. Washing with a 1 to 5,000 corrosive sublimate solution, and the application of white precipitate ointment, is the best local treatment.

TINEA TRICHOPHYTINA

(Ringworm)

Ringworm is a contagious parasitic disease caused by a vegetable fungus, and capable of attacking any part of the body surface.

ETIOLOGY.—Two varieties of parasitic fungi have been identified as causative agents, the *microsporon Andouini* or small-spored fungus, and the *trichophyton,* or large-spored fungus.

Ringworm occurs so much more frequently in children than in adults, that it may be considered as essentially a disease of early life. It is acquired either from another child, or from one of the lower animals, particularly the cat. It is transmitted either by direct contact, or through the medium of caps, brushes, combs, towels, and similar articles.

TINEA CIRCINATA.—This form is due usually to the *trichophyton,* and is characterized by ring-shaped vesiculo-squamous areas upon the body surface. At first are seen one or several hyperemic scaly patches, about the size of a pea. These become circular, and about the periphery may be seen small papules or vesicles. The areas spread peripherally, at the same time healing in the center, so that ring-shaped patches are formed, about the size of a coin. They are pinkish-red areas, with elevated borders, the centers showing a fine desquamation. Sometimes patches are seen with several concentric rings; at other times the areas are circular, without central clearing.

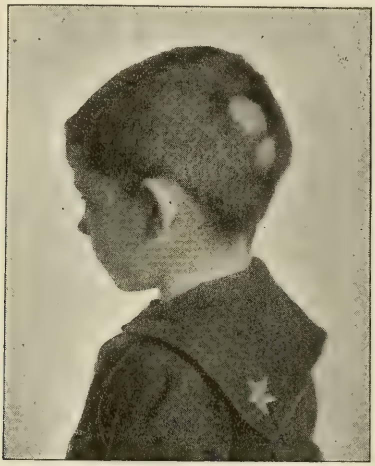
The face, neck, and backs of the hands are the most frequent seats of the disease. Itching is slight or absent.

TINEA TONSURANS.—In this form the disease involves the hair. It may be caused by either the *trichophyton,* or the *microsporon.* The disease begins on any part of the hairy scalp, with small reddened scaly patches. They occasionally have an annular, slightly elevated border. The process soon invades the follicles and hair shafts, and the hair becomes brittle, breaking off about half an inch above the level of the skin. Some of the hairs fall out entirely. The typical lesions are partly bald spots consisting of discrete, rounded, slightly reddened patches, with grayish scales. The areas may vary in size from that of a small coin up to that of the palm of the hand. The disease extends by the involvement of fresh hairs at the periphery of the lesion. The scalp may become diffusely infected without distinct circumscribed patches, but with a general thinning of the hair.

DIAGNOSIS.—Tinea circinata is easy of recognition Tinea tonsurans must be distinguished from eczema, psoriasis, seborrhea, and alopecia areata. The partial baldness of the patches, and the broken off stumps of hair, are important. The diagnosis may always be confirmed by finding the fungus under the microscope. Epidermal scales are scraped off with a knife, and placed on a slide with a drop of caustic potash solution (20%). A coverglass is applied, with enough pressure to flatten out the scales. The fungus is easy to recognize.

PROGNOSIS.—Tinea circinata responds rapidly to treatment. Tinea tonsurans is much more resistant; the prognosis is favorable as to ultimate cure, but most cases persist from two months to a year or more.

FIG. 272



Tinea tonsurans. Male, 8 years old

TREATMENT.—For tinea circinata, rubbing in antiparasitic ointment once or twice a day is all that is required. Precautions should be taken to prevent the communication of the disease to others. As parasiticides, mercury, sulphur, betanaphthol, resorcin, tar, and chrysarobin are all useful.

℞

Sulphur	ʒ ii
Betanaphthol	ʒ i
Acid Carbolici	ʒ ss
Vaseline	℥ i

For tinea tonsurans, the scalp must be thoroughly cleansed each day with soap and hot water, and the antiparasitic ointment rubbed in thoroughly. I prefer the same preparation as that given for tinea circinata. Equal parts of oil of cade and olive oil are also useful. The hairs in the diseased and immediately surrounding areas should be carefully depilated.

Treatment of tinea tonsurans with the Roentgen ray has been very successful. The treatment should be given by an expert roentgenologist.

SCABIES

(Itch)

ETIOLOGY.—Scabies is a contagious, animal parasitic disease caused by the *Acarus Scabei.* The disease is most common in the lower social strata. Close bodily contact, or infected clothing are the usual means of transmission. Scabies is a household disease, its spread being largely due to overcrowding. Children are frequently affected.

SYMPTOMS.—The itch-mite first forms at the point of entrance into the skin a small papule, vesicle, or pustule. The typical lesion produced by the parasite is the burrow, a grayish or brownish linear elevation of the epidermis, varying in length from 1/8 to 1/2 of an inch. At the end of the burrow may be found the acarus, a yellowish-white opaque object, just visible to the naked eye. The burrows are often difficult to find in infants and young children. They are found most frequently along the ulnar border of the hand, between the fingers, and upon the wrists.

In some cases secondary inflammatory lesions are comparatively few. In other cases, particularly in neglected children, or in children in poor physical condition, they are so severe as to hide the primary burrows. The secondary eruption is multiform, consisting of papules, vesicles, pustules, crusts, and excoriations due to scratching. Pustular eruptions are common in neglected infants.

The distribution of the lesions of scabies is characteristic, the areas most frequently affected being the interdigital spaces, the flexor surfaces of the wrists, the axillae, the toes (in infants), and in males, the genitals. The chest about the nipples, the abdomen about the umbilicus, and the buttocks are also frequently involved. The face is exempt, except occasionally in infants, when it is infected by a contact with the maternal breasts.

Itching is a constant and prominent symptom in scabies. It is apt to be more severe at night than by day.

DIAGNOSIS.—The diagnosis of scabies is rarely difficult. The finding of the characteristic burrows is diagnostic, but sometimes the secondary inflammatory lesions are so marked as to obscure the burrows. In such cases the diagnosis is based on the characteristic distribution of the lesions, and on the fact that several persons in one household are usually affected, particularly if they occupy the same bed.

PROGNOSIS.—There is little tendency to spontaneous cure, but the disease, no matter of what duration, is speedily curable under treatment.

TREATMENT.—Every night for three successive nights, a prolonged hot bath should be given with vigorous scrubbing with soap and a nail-brush. After the bath, all parts of the body below the neck should be anointed with the parasiticidal ointment, which must be thoroughly rubbed into the skin. In the morning the ointment is washed off, and the child dressed in clean clothes.

After three treatments some simple soothing ointment, such as those recommended for eczema, should be applied to the skin, until the secondary inflammatory lesions disappear.

The parasiticides most valuable against scabies are sulphur, beta-naphthol, balsam of Peru, tar, and styrax. It must be remembered that these are irritating to the skin, and cannot be used too strong, especially in children. The ordinary sulphur ointment is too strong for use in infants. For routine use the following may be employed:

℞

Sulphur Praecip	ʒ i
Beta Naphthol	ʒ i
Balsam Peruv	ʒ ii
Vaseline	℥ i

PEDICULOSIS CAPITIS

ETIOLOGY.—This condition is due to the invasion of the scalp by the pediculus capitis, or head-louse. The disease is most common in the public schools of the large cities. It is contracted by close contact, or by wearing infected head coverings, and is favored by uncleanliness and neglect of the scalp. It is much more common in girls than in boys.

SYMPTOMS.—The lice cause itching, and the consequent scratching causes excoriation of the scalp with exudation which dries in the form of crusts, matting the hair together. Papules, pustules, and excoriations are usually seen in the occipital region, or about the face and neck. A pustular dermatitis in the occipital region is very characteristic. The postcervical glands are often enlarged.

The ova of the pediculi, or "nits," are present in abundance. They are grayish, pear-shaped bodies, attached to the hair by a membranous sheath. The pediculi themselves may usually be found on careful search.

DIAGNOSIS.—The presence of the pediculi and "nits" is diagnostic. The nits may be distinguished from epidermal scales, in that they may be slid along the hair without becoming detached.

PROGNOSIS.—Favorable, with treatment.

TREATMENT.—While the most effective measure in treatment is to cut the hair short, this is not always necessary, although more time and labor is often required, if the hair is not sacrificed. If the condition is discovered before there is much matting of the hair and crusting of the scalp, the hair can be preserved. If the condition is advanced, cutting the hair is better.

The hair at night should be saturated with crude petroleum, and a skull-cap to include the whole hairy scalp should be applied. In the morning a thorough shampoo with soap and water should be given. This should be done for one or two nights, and then attention should be directed to removing the nits. Frequent applications of a borax solution, or of a dilute solution of acetic acid (Acidi acetici ʒii Aquae ℥viii should be made, and the nits removed with a fine-toothed comb The eczema rarely requires treatment, subsiding when the cause is removed.

ECZEMA SEBORRHEICUM

(Seborrheic Eczema). (Seborrhea Corporis)

This disease is sufficiently common among children to warrant description here. It is usually regarded by pediatrists as a manifestation of ordinary eczema, but the evidence points toward an entirely different etiology.

ETIOLOGY.—This disease is generally regarded as of parasitic origin. Unna and Elliott have described a microörganism which they believe to be the cause of the disease. If parasitic, the disease is probably very feebly contagious, and its development is probably favored by all conditions lowering the general resisting power of the skin.

SYMPTOMS.—There is usually an antecedent seborrhea of the scalp with fine scaling. In seborrheic eczema, the scaling increases, and reddish patches develop, with some loss of hair. The reddening of the scalp may be diffuse or circumscribed, and may extend beyond the hair upon the forehead. On the reddened areas are loosely attached, greasy scales.

The process next extends to the postauricular region, and then to the face, where the naso-labial fold is a common seat of the eruption. Yellowish-red, scaling patches are found. From these regions the process may extend to any part of the body, forming the same patches. The regions of the sternum and between the scapulae are favorite seats of the eruption.

Itching is usually absent, or very slight. There is little inflammatory reaction.

DIAGNOSIS.—This condition is distinguished from ordinary eczema by its origin upon the scalp, by the absence of marked itching, by the absence of vesicles, pustules, inflammation, or infiltration and by the greasy scales.

PROGNOSIS.—There is no tendency toward spontaneous cure. Recovery can be brought about by treatment, but the length of time required is very variable.

TREATMENT.—Apply to the scalp at night the following lotion:

℞

Resorcini	ʒ ii
Spirit. vini rect.	
Aquae	aa ℥ iv

Wash this off in the morning, and apply a sulphur ointment in the strength of one drachm to the ounce. On the face and body, only the sulphur ointment is required; it should be applied twice a day.

VERRUCAE

(Warts)

ETIOLOGY.—No specific microörganisms have been found as the cause of warts. Nevertheless, according to the preponderance of the evidence, warts are contagious and autoinoculable. They are regarded as probably caused by microörganisms.

SYMPTOMS.—Warts consist of circumscribed elevations of the skin, due to hyperplasia of the papillae of the corium and the overlying epidermal layers. The common wart, seen upon the hands, is a rounded elevation about the size of a pea; the surface may be rough or smooth in texture, and yellowish or brownish in color. This is the only form common in children.

TREATMENT.—For common warts on the hands in children, treatment with caustics is the best method. The best caustics are glacial acetic acid, nitric acid, caustic potash, and chromium trioxide. These should be cautiously applied at intervals of several days, until the wart disappears. I prefer glacial acetic acid, applied with the tip of a common parlor match. It may be used a little more frequently than the other caustics.

SEBORRHEA

ETIOLOGY.—There is much diversity of opinion as to the etiology of seborrhea. One view regards it as of parasitic origin, classing it with seborrheic eczema. Another view regards it as a functional disease of the sebaceous glands. The only form to be considered here is the seborrhea of the scalp seen in infants.

SYMPTOMS.—The disease is characterized by the formation on the scalp of crusts of a soft, greasy consistency, and a dirty yellow color. In neglected cases the entire scalp may be covered with a dense crust. Upon removal of the crusts, the underlying scalp usually appears healthy, but if the case has existed a long time, or if efforts have been made to remove the crusts with a comb, a secondary eczema may develop.

DIAGNOSIS.—The condition is distinguished from seborrheic eczema, by the fact that crusting is marked, and out of all proportion to the reddening of the scalp, and by the absence of a tendency to extend downward onto the face.

PROGNOSIS.—There is very little tendency toward spontaneous improvement. Even under treatment the disease may last for months.

TREATMENT.—The crusts should be softened over night with olive oil, and removed by thorough washing with warm water and soap. The following ointment should be kept applied:

℞

Sulphur Precip....................	ʒ i
Lanolin..............................	℥ i

The softening and washing should be repeated as often as the crusts form.

DIVISION XVI

DISEASES OF THE NERVOUS SYSTEM

GENERAL ETIOLOGY AND CLASSIFICATION

The diagnosis and treatment of the diseases of the Nervous System has become a specialty requiring much expert knowledge, and many special methods of investigation. It would be impossible, within the limits of this book, to give a complete description of the nervous diseases encountered in early life. Nevertheless the pediatrist, and all who practice among children, must be prepared to encounter and recognize many cases of disease of the nervous system, and must be familiar with the peculiar manifestations of such disease when it appears in early life. I shall endeavor to present the subject in a way which will be useful to the pediatrist and to the general practitioner, rather than in the more complete way which would be required by the specialist in diseases of the nervous system.

The classification and description of the diseases of the nervous system is not easy. I have in this book adopted an etiological basis for the classification of diseases, because it is the most scientific basis, and the one which is most closely connected with the all-important questions of prognosis and treatment. The objection to an etiological classification for purposes of text-book description is the frequent lack of a definite relationship and correspondence between causes and symptoms. This difficulty is particularly marked in the diseases of the nervous system. In the first place, the symptoms produced by disturbances of function without organic lesions, with multiple causes, are much the same in their general character as the symptoms produced by organic lesions from such definite causes as trauma, infection, new growth, and congenital malformation. Furthermore, the nervous system is a highly specialized tissue, and its functional reaction to the causes of disease often bears a closer relationship to the *part* of the nervous system injured than it does to the *nature* of the injury. In other words, the symptoms of disease of the nervous system when there are organic lesions, depend more upon the *location of the lesion,* than upon the *particular nature of the cause.* It is for this reason that most text-books classify the diseases of the nervous system upon a purely anatomical basis, grouping the various processes according to the areas and tracts involved, rather than according to etiological factors. Such a method of classification has given us many terms, such as cerebral paralysis, bulbar paralysis, spinal paralysis, spastic paraplegia, each of which refers to a fairly characteristic clinical picture, but which may represent several different pathological processes each with a different etiology. The clinical descriptions corresponding to these terms are so familiar, and the terms themselves are in such general use, that they cannot be wholly neglected, even if they cannot be used for the names of diseases under an etiological classification. Their relationship to the various pathological processes must be clearly set forth.

The functional specialization of the various parts of the nervous system makes the topographical diagnosis of its lesions particularly fascinating. Neurologists have been able to carry the *localization* of nervous system lesions from the symptoms to a high degree of attainment, and have devoted much study to the devising of special tests of nervous function directed at the perfecting of topographical diagnosis. I sometimes think that a disproportionate amount of attention has been devoted to this aspect of the subject. Topographical diagnosis is not the only consideration, and if its perfecting has led to a tendency to minimize the importance of etiology, it is to be regretted.

There are two aspects to be considered in the diagnosis of a disease of the nervous system. One is the question of the *localization* of the lesion or disturbance, the other the question of the *nature and cause* of the lesion or disturbance. The first aspect is important mainly from the light which it throws upon the all-important second aspect. The physician must collect and review all the clinical evidence bearing on localization, and having done so, should decide where the trouble lies. This is only the first step in diagnosis. I have, however, observed a tendency to rest content with this, to consider that the diagnosis is made if, for instance, the lesion can be located definitely in the cortex, pons, cerebellum, spinal cord or peripheral nerves. The classification of the diseases of the nervous system upon an etiological basis in spite of the difficulties attending clinical description under such a method of classification, is a constant reminder that the diagnosis is not completed with localization, and that the second, most important step in diagnosis remains to be taken. This step is the recognition of the nature and cause of the pathological process.

The etiological factors in the diseases of the nervous system are the same as those of disease in general, namely: (1) The normal lack of development of early life; (2) inherited constitutional peculiarities; (3) unknown internal causes; (4) mechanical, chemical, or thermal injury from without; (5) external factors in hygiene and environment; (6) infection.

The ways in which these various causes produce disease are the following: (1) By producing faulty anatomical development in intrauterine life; (2) by producing primary anatomical lesions in extrauterine life; (3) by producing disturbance of function without anatomical lesions.

The following is the grouping of diseases of the nervous system which will be here employed:

I. Congenital Malformations and Developmental Anomalies.
  1 Meningocele, Encephalocele, and Hydrencephalocele
  2. Spina Bifida
  3. Defective Cerebral Development
  4. Mongolian Idiocy
II. Mechanical Injuries from External or Internal Causes.
  1. Traumatic Injuries
  2. Concussion
  3. Insolation
  4. Cerebral Hemorrhage
  5. Cerebral Thrombosis
  6. Cerebral Embolism
  7. Facial Paralysis
  8. Compression Myelitis
III. New Growths.
  1. Intracranial Tumors
  2. Tumors of the Cord
IV. Functional Disturbances.
  1. Convulsions
  2. Epilepsy
  3. Hysteria
  4. Chorea
  5. Spasmodic Neuroses
  6. Retarded Psychical Development
  7. Retarded Speech
  8. Headaches
  9. Neuralgia
  10. Disturbed Sleep
  11. Pavor Nocternus
V. Infections.
  1. Acute Meningitis
  2. Tuberculous Meningitis
  3. Encephalitis
  4. Abscess of the Brain
  5. Septic Sinus Thrombosis
  6. Solitary Tubercle
  7. Cerebral Syphilis
VI. Organic Diseases of Unknown or Miscellaneous Causes.
  1. Hydrocephalus

2. Pachymeningitis
3. Amaurotic Family Idiocy
4. Hereditary Ataxia
5. Insular Sclerosis
6. Syringomyelia
7. Hereditary and Familial Spastic Paralysis
8. Multiple Neuritis
9. Progressive Muscular Atrophy

I. CONGENITAL MALFORMATIONS AND DEVELOPMENTAL ANOMALIES

Many of the malformations of the brain, of which there are a great variety, are incompatible with life, and are only of anatomical interest. Only those varieties will be described which are of clinical interest. Malformations of the spinal cord are frequently associated with those of the brain, and most of them are rare, and of very little practical interest.

MENINGOCELE, ENCEPHALOCELE AND HYDRENCEPHALOCELE

These names are given to three congenital malformations, which differ in their anatomy, but in all of which there is a protrusion of some part of the cranial contents through an opening in the skull.

ETIOLOGY.—The ultimate etiology of these lesions is that of the congenital malformations in general, and is wholly unknown. It is probable that they are caused by an anomaly of fetal development, rather than by intrauterine disease of tissues already properly formed. The cause of these developmental anomalies is, and probably always will be, one of the mysteries of medicine. The most plausible theory of the immediate pathogenesis of encephalocele and kindred lesions, is that the primary malformation results in an intrauterine hydrocephalus, and that as the development of the bones gradually encloses the cranial cavity, a portion of the contents is left outside.

PATHOLOGICAL ANATOMY. MENINGOCELE.—In this condition, which is the rarest form, there is a protrusion of the membranes alone through a hole left in some part of the cranial wall by defective ossification. The tumor consists in a sac, which is usually distended with cerebrospinal fluid.

ENCEPHALOCELE.—In this condition the protruding sac contains some portion of the brain substance, which is connected with the rest of the brain by a constricted pedicle. The tumor may or may not contain fluid; if fluid be present, it lies between the membranes and the protruding cerebral substance. This malformation is sometimes called *hernia cerebri*, and is more common than meningocele.

HYDRENCEPHALOCELE.—In this condition there is a protrusion not only of a part of the brain, but also of a part of the cerebral ventricles. The protruding part of the brain contains a cavity which is filled with cerebrospinal fluid, and which communicates with the lateral ventricles through a narrow opening in the pedicle of the tumor. This condition is undoubtedly due to a fetal internal hydrocephalus, and is the most common malformation of the three.

All these conditions are comparatively rare. They are frequently associated with other congenital developmental anomalies, such as hare-lip, cleft palate, club-foot, and spina bifida.

The opening of the cranium may be in either the occipital or in the frontal region. It is usually in the median line, but may be in other situations, along the lines of any of the sutures. An occipital opening may be at the posterior fontanel, in the middle of the occipital bone, or may communicate with the foramen magnum. A frontal opening may pass between the lateral halves of the frontal bone, or between the cribriform plate of the ethmoid and the frontal bone. In the latter situation, called the naso-frontal form, the tumor is usually a little to one side of the median line. Rarely the opening allows the tumor to project into the pharynx.

SYMPTOMS.—The tumor is always present at birth, although later it may show a notable increase in size. In shape it is round or pyriform; it may or may not be pedunculated. The surface is smooth or lobulated. The size varies from that of a hazel nut to that of a child's head, the ordinary size being about that of a tennis ball. The tumor is usually covered by the scalp, which is often devoid of hair; at times it may be covered only with granulation tissue, or it may show a central cicatrix. On palpation the tumor is elastic, and usually pulsates synchronously with the heart. It is usually fluctuating. It increases in size and tension during crying. It is usually capable of partial, and occasionally of complete reduction, but efforts at reduction are usually accompanied by marked cerebral symptoms, even convulsions.

In *meningocele* the tumor is at first small in size, but increases; it is smooth, pedunculated, and distinctly fluctuating; it is often completely reducible, but compression causes cerebral symptoms; it rarely pulsates. The most important point in its diagnosis is its complete translucency.

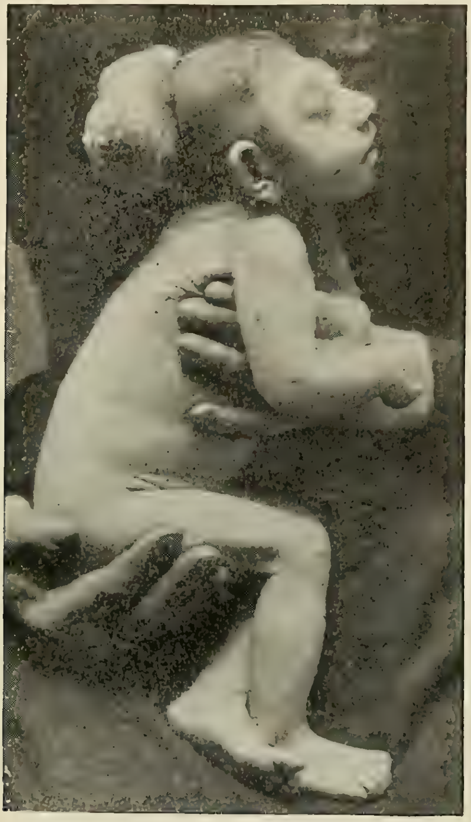
In *encephalocele* the tumor is small, and does not increase markedly in size; it is smooth, and rarely pedunculated; it is not fluctuating; it is usually reducible, and pressure causes cerebral symptoms; there is pulsation; it is not translucent. The most important points are the absence of translucency and fluctuation.

In *hydrencephalocele* the tumor is large, pendulous, and pedunculated; the surface is irregular or lobulated; it is distinctly fluctuating; it is irreducible, and pressure rarely causes symptoms; there is pulsation; it is sometimes partly, but rarely completely, translucent.

The most important points are the large size, irregular shape, impossibility of reduction, fluctuation, and absence of complete translucency.

DIAGNOSIS.—The only conditions giving swellings upon the scalp in newborn infants are *caput succedaneum*, and *cephalhematoma.* The soft, flabby, non-fluctuating swelling of the former is not likely to be confounded with these malformations. The fluctuation of cephalhematoma might cause it to be mistaken for meningocele or hydroencephalocele. In cephalhematoma the tumor is never pedunculated, never reducible, pressure does not cause symptoms, it never pulsates, it is never translucent, and it rarely occupies the median line.

FIG. 273



Female, 2 months old. Hydroencephalocele

PROGNOSIS.—The outlook is not very favorable, most cases dying in the early weeks of life. The occipital tumors are more serious than the frontal. In spite of care, rupture and infection is likely to take place, death occurring as the immediate result of the rupture, or from meningitis or convulsions. Spontaneous cure, with shutting off of the tumor, may occur in meningocele before rupture occurs. In encephalocele, the tumor grows little or not at all, and rupture is not so likely to occur, but the patients usually show various symptoms of organic brain disease. The prognosis of hydrencephalocele is absolutely bad, with or without treatment.

TREATMENT.—This is unsatisfactory. Meningocele has been treated by simple aspiration, by ligature of the pedicle after dividing the skin, and by a plastic operation designed to close the opening. While all these procedures have at times been successful, recovery has often been followed by the development of hydrocephalus. As spontaneous cure sometimes occurs, I believe it best to treat even meningocele by protection and slight compression, using aspiration only if the tumor becomes notably tense, or if rupture is threatened. In encephalocele, protection and compression are all that should be attempted. In hydrencephalocele the prognosis is so absolutely bad, that the question of operation may be considered, but it affords little prospect of success.

SPINA BIFIDA

This is a congenital malformation consisting in a lack of closure of the spinal canal with a protrusion of some part of its contents.

ETIOLOGY.—The ultimate etiology of this condition is that of the congenital malformations in general, and is wholly unknown. It is certain that the malformation in spina bifida is purely developmental in origin, for there is evidence that it takes place at a very early period of fetal development. The anatomy of the lesion varies according to the period of development at which the malformation occurs. In some cases it occurs at an early period of fetal life, before the cord becomes separated from the epiblastic layer from which it is developed. In such a case the cord remains adherent to the epiblastic covering, and the mesoblastic structures which should be formed between the cord and the skin, such as the vertebral arches, muscles, and inner layers of the integument, remain undeveloped. In this condition the wall of the sac contains the elements of the cord, nerves and meninges, which are intimately fused together. When the developmental fault occurs later, there is a similar lack of development of the mesoblastic structures, but the cord and nerves, while attached to the sac, are not so intimately fused with it. In still other cases it is only the meninges which enter into the formation of the sac, the cord remaining within the spinal canal. In this last condition, the failure of closure of the laminae of the vertebrae is probably due to pressure from the accumulation of fluid within the sac.

PATHOLOGICAL ANATOMY. SPINAL MENINGOCELE.—In this form the sac consists of the membranes only. It is filled with cerebrospinal fluid. The communicating opening between the tumor and the spinal canal is small, usually not more than 1/12 to 1/4 of an inch in diameter. In some cases the communicating opening is closed, the sac having become shut off by the development of the intervening structures. The tumor is usually globular and is sometimes pedunculated. It varies very greatly in size, but may become as large as five or six inches in diameter. It is elastic, compressible, fluctuating, and, unless the communicating opening be closed, it is increased in size and tension by crying. The skin over the tumor in meningocele is usually fully developed. Spinal meningocele is most frequently seen in the cervical region, but may occupy the umbo-sacral-region, as do the other forms of spina bifida. It is a comparatively rare form.

MENINGOMYELOCELE.—This form of spina bifida is very much the most common, and is the variety usually meant when spina bifida is spoken of. It is usually seen in the sacro-lumbar region. The accumulation of fluid, instead of taking place in the posterior arachnoid space as in meningocele, takes place in the anterior subarachnoid space. For this reason the cord is contained in the protruding sac usually forming a part of its wall. The relation of the spinal cord to the wall of the sac in these tumors is a very variable one. The cord may run horizontally along the top wall of the tumor to the central cicatrix with which it becomes blended, and from which the spinal nerves run back along the lower wall of the tumor, re-entering the spinal canal and being distributed as usual. In some cases the cord may be suspended from the wall of the sac by a sort of mesentery. Most commonly, however, the cord joins the wall of the sac soon after its entrance through the opening in the spinal canal, and the fibers of the cord are spread out all over the surface of the wall of the sac like a fan, coming together again below, where they re-enter the spinal canal.

The tumor in meningomyelocele is smaller than in spinal meningocele, the usual size being that of a tennis ball. It is never pedunculated. As in all forms of spina bifida, it is elastic, compressible, fluctuating, and is increased in size and tension by crying. It is usually only partly covered by skin, having a central area of variable size where there is only a thin translucent membrane. This membranous surface, usually elliptical in shape, is known as the central cicatrix. It may be more or less covered with granulations, and often shows a tendency to ulcerate. In the center there is often a sort of umbilical depression corresponding to the original point of fusion between the cord and the membranes.

SYRINGOMYELOCELE.—This is very much the rarest form of spina bifida. It may be found higher up, in the dorsal or dorso-lumbar region as well as in the lumbo-sacral region. The accumulation of fluid is in the central canal of the cord, the condition corresponding anatomically to the hydrencephalocele of the cranium. The lining of the sac in this form consists in the attenuated and atrophied elements of the cord. This form is usually associated with hydrocephalus, and has the worst prognosis.

Although in most cases these malformations are associated with a bifid spine, this is not always the case. The protrusion in rare instances may be through the intervertebral notch, or even anteriorly between the bodies of the vertebrae, the tumor projecting into the thorax, abdomen, or pelvis. These anomalies are so rare as to be merely anatomical curiosities. At times there may be a bifid spine but the tumor may be so small as to be unrecognizable externally. This last condition is known as *spina bifida occulta*, and has been supposed to account for certain obscure symptoms of disturbance of the nervous system. It can only be recognized by roentgenograms of the spine.

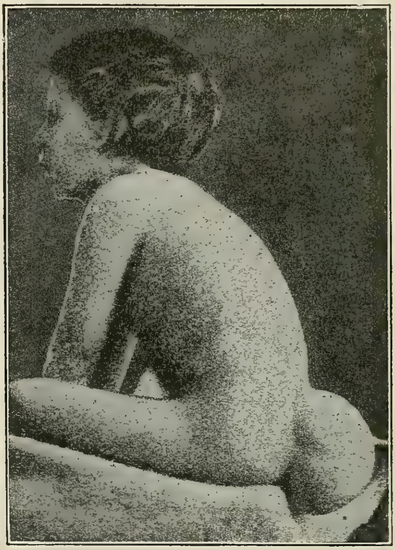
Spina bifida is very frequently associated with other malformations, such as hare-lip, cleft palate, club-foot, hydrocephalus, cerebral meningocele, and so forth.

SYMPTOMS.—The tumor in spina bifida is always congenital. Its characteristics have been described under pathological anatomy. In spinal meningocele there are no symptoms other than the presence of the tumor.

In the other forms, meningomyelocele and syringomyelocele, paralysis is usually present. The degree and extent of the paralysis varies with the extent to which the cord is involved in the malformation. In the most marked cases there is complete paraplegia with paralysis of the bladder and rectum. When the tumor is low down in the lumbo-sacral or sacral region, only the cauda equina may be involved in the malformation, and this to a variable extent, so that paralysis of the extremities may be incomplete, and the bladder and rectum may escape. Rare cases have been observed in which spina bifida occulta was responsible for obstinate incontinence of urine, and sometimes also of feces, without paraplegia.

The tumors in spina bifida tend to increase steadily in size. If the tumor is covered by skin, as is usually the case in spinal meningocele, the growth of the tumor may be almost unlimited. If, as is usually the case in the common form, the skin is lacking, the wall of the sac is so thin that rupture is almost certain to take place in the course of the first few months of life, either spontaneously or through some accident. When rupture occurs the usual immediate result is convulsions and marked signs of cerebral disturbance from the rapid draining of the cerebrospinal fluid, and death may occur from this, or may occur from the infection which inevitably takes place after rupture. Infection and meningitis may also take place without rupture of the sac, the microörganisms passing through its wall. In rare cases infection of the sac has resulted in a cure, the inflammation in the wall of the sac resulting in the closure of the small opening into the spinal canal with sloughing and cicatrization of the parts left outside. Usually, however, infection of the sac is rapidly followed by spinal meningitis, which soon extends upward to the brain. Many cases die as a result of malnutrition before rupture and infection occur.

FIG. 274

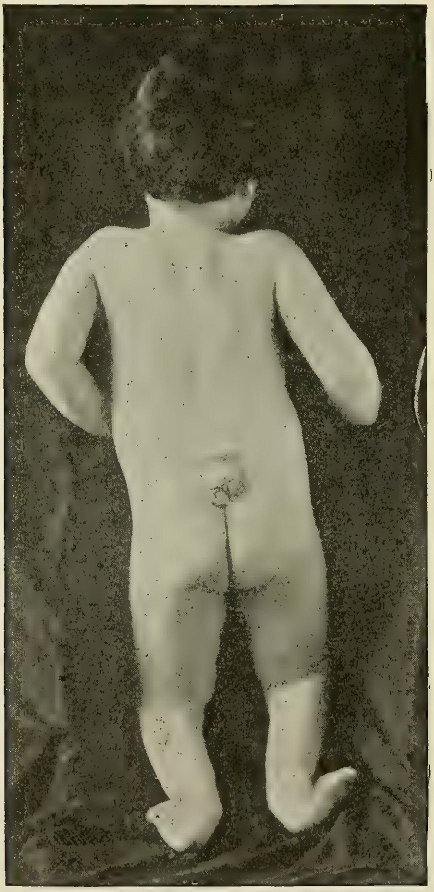


Spina bifida of lumbar region. Male, 5 years old.

DIAGNOSIS.—There is no difficulty in the recognition of spina bifida. No other condition gives a congenital fluctuating tumor in this situation. It is sometimes more difficult to distinguish between the various anatomical forms, although such distinction is very important in prognosis and treatment. Spinal meningocele is recognized by the absence of a palpable fissure in the spine, the presence of a pedunculated tumor covered by the skin, the presence of complete translucency, and the absence of paralysis. Meningomyelocele is recognized by the presence of a sessile tumor with a membranous central cicatrix, a palpable bony fissure, and the presence of a variable degree of paralysis. The coexistence of hydrocephalus suggests syringomyelocele.

PROGNOSIS.—This depends upon the anatomical variety which is present and upon the extent of involvement of the spinal cord in the malformation. The prognosis is best in simple meningocele covered by integument; in this form complete recovery frequently occurs, sometimes spontaneously, more often as a result of operation. In meningomyelocele with complete paraplegia or with extensive paralysis, the prognosis is bad; if there is only a limited paralysis in the extremities and no paralysis in the rectum or bladder, the prognosis is better, although the condition is always a serious one. In many cases in which operation has resulted in a successful cure of the spina bifida, hydrocephalus has subsequently developed. I have recently had a case in which there was infection of the sac without meningitis which resulted in a spontaneous cure of the spinal malformation, but in which the cure was followed by the develop-

FIG. 275



Spina bifida. Spontaneous cure. Male, 4½ years old ment of hydrocephalus. There is always danger of a fatal ending from malnutrition, rupture of the sac, or meningitis. The prognosis in cases of syringomyelocele with hydrocephalus is hopeless.

TREATMENT.—The first essential of treatment is completely to protect the tumor from pressure. This can be accomplished by surrounding the tumor with a large pad of absorbent cotton, or still better, having the child lie upon a rubber-ring cushion. When the tumor is not covered by skin, it must be kept absolutely clean. It should be covered with some drying powder, and should only come in contact with absolutely sterile materials.

Further treatment is surgical. The old treatment of injection of the sac was not followed by good results and has now been entirely given up. The treatment now employed consists in excision of the sac. The matter is a complicated one on account of the usual involvement of the cord to a greater or less extent, and for the details of the various plastic operations which have been proposed, the reader is referred to works on operative surgery. The time of operation depends upon the nature of the tumor and upon how rapidly it is growing. If the tumor is covered by the skin, it is best to wait until the child is at least six months old. If the tumor is uncovered by skin and growing slowly, it is best to wait only until nutrition has become well established. If the tumor is growing fast and there is evident risk of spontaneous or accidental rupture, it is dangerous to wait. Contraindications to operation are complete paraplegia with involvement of the rectum and bladder, hydrocephalus, and extreme malnutrition.

In meningomyelocele, although recovery may follow operation, it is only rarely complete. In the majority of cases a varying degree of paralysis in the lower extremities is left with the resulting muscular atrophy, contractures, and deformities. Hydrocephalus often develops after the cure, and complete recovery is seen most often in spinal meningocele.

DEFECTIVE CEREBRAL DEVELOPMENT

(Idiocy; Imbecility; Mental Deficiency; Feeble Mindedness)

This is a congenital condition of developmental origin characterized by some gross anatomical lesion of the brain.

The terms given above as synonyms for defective cerebral development are not really synonyms. Mental deficiency, idiocy, imbecility, and feeble mindedness are terms representing a varying degree of severity of a fairly constant clinical picture. From the etiologic point of view, however, mental deficiency is only a symptom seen in a variety of cerebral lesions, and the clinical manifestations may be the same whether the origin of the lesion be a congenital developmental anomaly or some lesion acquired in extra-uterine life. This condition illustrates one of the difficulties attending the classification of diseases upon an etiological basis. It is much simpler to describe the clinical manifestations seen in mental deficiency, and then to enumerate the various causes which may produce this condition. Nevertheless the most typical cases of mental deficiency are due to congenital developmental anomalies, and for this reason the condition is best described under this etiological heading. Other conditions which from the symptomatic point of view have been described as separate diseases, also appear under defective cerebral development as particular manifestations. Among these conditions is one which has been called by various names, such as *cerebral paralysis of intra-uterine origin,* congenital spastic diplegia, paraplegia, or hemiplegia. *Microcephalus* also is usually described as a separate disease from the clinical standpoint. All of these conditions belong in the same etiological group.

ETIOLOGY.—All that is positively known of the etiology of defective cerebral development is that the lesion is produced during intra-uterine life. The ultimate causes are those of the congenital malformations in general, which are wholly unknown. It has been fairly well established that congenital malformations may arise in two ways, first, from a fault in fetal development affecting the formation of the tissues, and second, from intra-uterine disease affecting tissues already formed. In conditions of defective cerebral development, the relation of the various lesions found to known facts in embryology is less definite and clear than in many other varieties of malformation. It is therefore impossible to know how great a part is played by a purely developmental anomaly on the one hand, and by intra-uterine disease on the other. It is quite probable that in some cases one factor is operative, in other cases the other, and in still other cases both combined.

It is known, however, that there is in many cases of defective cerebral development a strong hereditary influence. This shows itself not only in the existence of a similar condition in the parents or in other members of the same generation, but also at times in the existence in the parents of other forms of disturbance of the nervous system, such as epilepsy, hysteria, and the various forms of insanity. At times there is a history of syphilis or alcoholism in the parents, which is strongly suggestive of intra-uterine disease as the chief factor in the production of the defective development of the brain. Sometimes there is a history of consanguineous marriage. At all events heredity is the only important etiologic factor of which there is any definite evidence. It is possible also that a great variety of conditions beside alcoholism and syphilis, which may affect the health of the mother during pregnancy, may be factors in producing a defective development of the brain of the offspring.

PATHOLOGICAL ANATOMY.—The pathological changes which are found in the brains of defectives show a great variety both in character and in degrees of severity. There may be a defective development of any portion of the brain, which is clearly visible macroscopically. The affected portions show a greater or less degree of atrophy, or one or more lobes may show failure of development, or there may be portions of the cortex of variable extent which show poorly developed convolutions and shallow sulci. A detailed enumeration of all the anatomical conditions found in these congenital anomalies is not of practical importance. One or two, however, deserve particular mention.

In some cases no pathological changes can be discerned macroscopically. In this condition, which has been described under the term *agenesis corticalis,* the size, weight and macroscopic appearance of the brain are normal, but the microscope shows a more or less complete arrest of development of the nerve cells of the cortex. When this condition involves the motor areas, it can be readily seen how congenital spastic paralysis would result.

In some cases the lesion is what has been described as *porencephalus.* There is an acquired form of porencephalus which is one of the late results of meningeal hemorrhage, but the congenital form is one of the lesions associated with the symptom complex of defective cerebral development. The lesion consists in a large depression in some part of the brain with the surrounding parts well developed. The hole may involve the whole lobe and may be deep enough to reach the lateral ventricles.

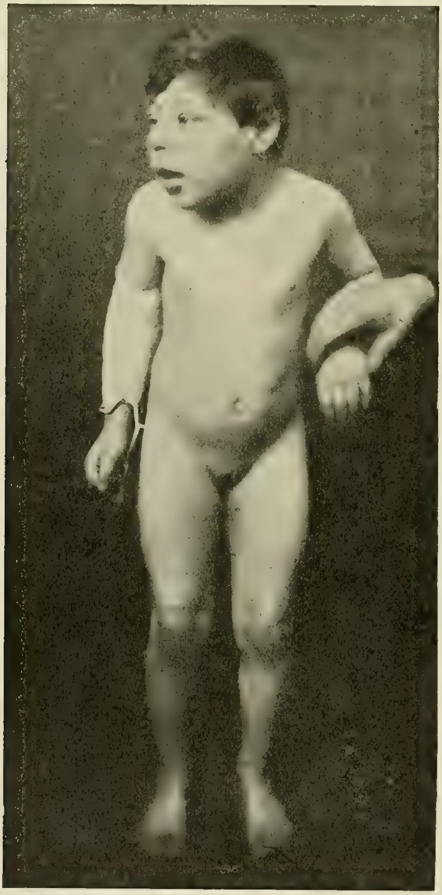
MICROCEPHALUS.—This is the particular lesion which is best known in connection with the developmental anomalies of the brain, and under a classification upon an anatomical basis it is usually regarded as a separate disease. The lesion consists of an arrested growth of the brain with a premature ossification of the skull, the result being a head which is much smaller than the normal. Microcephalus has often been regarded as being due to premature ossification of the skull, the arrested development of the brain being secondary. It is probable, however, that this view is entirely wrong, and that the primary lesion is a defective development of the brain, to which premature ossification of the skull is secondary. This view explains why the operation of craniectomy, once thought promising in the treatment of microcephalus, has been abandoned.

SYMPTOMS.—The principal symptoms of defective cerebral development are:

1. Mental deficiency
2. Spastic paralysis

Either of these conditions may exist alone or both may be combined. If the lesion involves only the motor areas of the brain, the result will be some form of congenital cerebral paralysis without mental deficiency. If, on the other hand, the lesion spares the motor areas of the brain, the result will be a varying degree of mental deficiency without spastic paralysis. Many defectives are entirely without stigmata suggesting disease of the upper motor tracts. On the other hand, in many other defectives the coexistence of a spastic paralysis is one of the most important points in the diagnosis of the cause of the defective mentality. The symptoms of mental deficiency and of congenital cerebral paralysis will be described separately, although it must be remembered that they are often combined.

FIG. 276



Microcephalic idiot, 10 years old

MENTAL DEFICIENCY.—There are all grades of mental deficiency. The term *idiocy* is applied to a condition in which mental development is never beyond that of the normal child of two years. The term *imbecility* is applied to a slightly higher grade in which the degree of mentality finally attained is not beyond that of the average child of seven years. The term *moron* is applied to children whose mentality does not reach a higher degree than that of average children of twelve years. There are still milder grades of mental deficiency which, however, cannot be recognized in children, but which are recognized by certain peculiarities of psychic development which manifest themselves in young adult life. These cases are spoken of as *high grade imbeciles.*

The description of all these different grades of mental deficiency would occupy more space than can be allotted to it in a general textbook. I shall content myself with a description of typical cerebral idiocy.

The symptoms of idiocy vary according as the individual represents a high or a low grade of this condition. An idiot may have a large head from hydrocephalus, or a small head from microcephalus. Again, idiots may have normally developed crania both as to size and as to shape. In the lower grades there is often some physical malformation in connection with the mental impairment. The temperament may be violent or good-natured, the mood often alternating. The sense of morality is wholly lost. Extreme mischievousness or even cruelty to animals and other children may be an early indication of the perverted nature of the child. In the more severe cases of idiocy there is considerable incoördination of the limbs, and the movements of the child are awkward and irregular. In many cases the speech is almost unintelligible. The idiot does not take notice of surrounding objects as does the normal child, and even when the sight and hearing are perfectly normal the impressions made on the senses are deadened. Epileptiform convulsions very commonly accompany idiocy, and play a most important part in the general condition of the patient.

The symptoms which are usually met with, and which enable us to diagnose a pronounced case of idiocy, are the characteristic expression, the occasional presence of strabismus, the drooping head, the drooling, and the lack of all idea of cleanliness. Sometimes the child is so limp that he is unable to bear his weight at all, or will stand held by the hands, with his feet apart, his knees bent, and his trunk leaning forward. The whole body sways to and fro with an oscillating movement and absence of equilibrium. When able to walk alone he walks in a staggering, uncertain way, and falls easily. In many cases, however, the child cannot even sit up alone. The muscles of the neck are often so weak that the head falls over on one shoulder or forward on the chest. The vertebral column fails to support the trunk and bends to a marked degree, and all the muscles are feeble and comparatively useless. Lack of power of attention and lack of memory exist in all cases, and in the higher grades are often the most prominent symptoms.

Milder grades can only be recognized by a comparison between the mental development of the patient and that of an average child of the same age. It is, however, an essential feature in the clinical picture of a defective that he is not merely backward but *abnormal.* His condition never is that of a normal child of a younger age, but as he becomes older the abnormal characteristics of his character and mental processes become more and more apparent. Prolonged observation is often required to distinguish between simple backward development and mental deficiency. Specialists in the subject have developed various tests by which the mental capacity of children can be estimated and standardized. This, however, is a matter for the specialist, as is also the training of these defectives. The pediatrist and general practitioner, however, must be in a position to recognize the abnormal, to distinguish mental deficiency due to organic disease from simple retarded development, and to trace mental deficiency to its cause.

In judging the mental development of a given child in reference to its age one will find the following summary of the normal state of development in the early months of life very helpful. It is taken from Church and Peterson's "Nervous and Mental Diseases," and was abstracted by them from Pryer's, "The Mind of the Child."

First Month.—Sensitive to light as early as first and second days. Pleasure in light of candle and in bright objects on eleventh day. Hears on fourth day. Discriminates sounds last two weeks of month. Starts at gentle touches second and third days. Sensibility to taste about end of first week. Strong-smelling substances produce mimetic movements at birth. Pleasure first days in nursing, in bath, in sight of objects. Discomfort first days from cold, wet, hunger, tight clothing. Smiles on twenty-sixth day. Tears on twenty-third day. Vowel-sounds in first month. Memory first active as to taste and smell, then as to touch, sight, hearing. Incoördinate movements of eyes. Sleeps two hours at a time, and sixteen hours in twenty-four. Reflexes active.

Second Month.—Strabismus occasionally until end of month. Recognizes human voices; turns head toward sounds. Pleased with music and with human face. Sleeps three, sometimes five or six hours. Laughs from tickling at eighth week. Clasps with its four fingers at eighth week. First consonants from forty-third to fifty-first days (am-ma, ta-hu, go, ara).

Third Month.—Sixty-first day, cry of joy at sight of mother and father; eyelids not completely raised when child looks up. Accommodates at ninth week. Notes sound of watch at ninth week; listens with attention.

Fourth Month.—Eye-movements perfect. Objects seized are moved toward the eyes. Grasps at objects too distant. Joy at seeing self in mirror. Contraposition of thumb in grasping at fourteenth week. Head held up permanently. Sits up with back supported at fourteenth week. Beginning to imitate.

Fifth Month.—Discriminates strangers. Looks inquiringly; pleasure in crumpling and tearing newspapers, pulling hair, ringing a bell. Sleeps ten to eleven hours without food. Desire shown by stretching out arms. Seizes and carries objects to mouth. Consonants l and k.

Sixth Month.—Raises self to sitting posture. Laughs, and raises and drops arms when pleasure is great. "Crows" with pleasure. Compares image of father in mirror with original.

Seventh Month.—Astonishment shown by open mouth and eyes. Recognizes nurse after four weeks' absence. Sighs. Imitates movements of head, of pursing lips. Averts head as a sign of refusal. Places himself upright on lap.

Eighth Month.—Astonishment at new sounds and sights; at imitations of cries of animals.

Ninth Month.—Stands on feet without support. More interest shown in things in general. Strikes hands together with joy. Shuts eyes and turns head away when something disagreeable is to be endured. Fear of dog. Turns over when laid face downward. Turns head to light when asked where it is. Questions understood before child can speak. Voice more modulated.

Tenth Month.—Sits up without support in bath and carriage. First attempts at walking at forty-first week. Beckoning imitated. Missed parents in absence, also a single nine-pin of a set. Cannot repeat a syllable heard. Monologues and hints at imitation (ma, pappa, tatta, appapa, baba, tata, pa, rrrr, rrra).

Eleventh Month.—Screaming quieted by "sh." Sitting becomes habit for life. Stands without support. Stamps. Syllable correctly repeated. Whispering begins. Consonants b, p, t, d, m, n, r, l, g, k, vowel a most used, u and o rare, i very rare.

Twelfth Month.—Pushes chair. Can not raise self or walk without help. Obeys command. Gives the hand.

Thirteenth Month.—Creeps. Shakes head in denial. Says papa and mamma. Understands some words spoken.

Fourteenth Month.—Can not walk without support. Raises himself by chair. Imitates coughing and swinging of arms.

Fifteenth Month.—Walks without support. Laughs, smiles, gives a kiss on request. Repeats syllables. Understands ten words.

Sixteenth Month.—Runs alone. Falls rarely.

Seventeenth, Eighteenth, and Nineteenth Months.—Sleeps ten hours at a time. Associates words with objects and movements. Blows horn, strikes with hand or foot, gives leaves to stag, waters flowers, puts stick of wood in stove, washes hands, combs and brushes hair, and other imitative movements.

Twentieth to Twenty-fourth Months.—Marks with pencil on paper, whispers in reading newspaper. Very few expressions of his are recognizable. Executes orders with surprising accuracy. Tries to sing and beat time and dance.

Twenty-fifth to Thirtieth Month.—Distinguishes color correctly. Sentences of several words. Begins to climb and jump and to ask questions.

Thirtieth to Fortieth Month.—Goes up-stairs without help. Sentences correctly applied. Clauses formed. Words distinctly spoken, but influence of dialect appears. Questioning repeated to weariness. Approximates manner of speech to that of family more and more.

SPASTIC PARALYSIS.—When this is seen as a symptom of congenital cerebral defect, it is of the same general type as is seen with acquired lesions involving the upper motor segment. Loss of power, while present, is not usually the most prominent symptom. The most marked symptom is usually a tonic spasm of the muscles. The extent of the paralysis is very variable. It is usually diplegic or paraplegic. While it may exist alone, it is usually combined with mental deficiency. The early diagnostic symptoms are paralysis, rigidity, and occasionally convulsions. There may be changes in the pupils and nystagmus. It must be remembered that at times the paralysis which accompanies defective cerebral development, instead of being of a rigid spastic character, consists in a general flaccidity of the muscles. This type is only seen in cases with pronounced mental deficiency.

DIAGNOSIS.—Defective cerebral development must be differentiated from other conditions characterized by deficient mentality, and also from other conditions characterized by cerebral paralysis. The conditions characterized by defective mentality are (1) idiocy from an acquired cerebral lesion; (2) cretinism; (3) mongolian idiocy; (4) amaurotic family idiocy, (5) retarded mental development.

Idiocy from an Acquired Cerebral Lesion.—As the symptom complex in the diseases of the brain depends more upon the anatomical lesions than upon cause, there is no essential difference in the clinical picture presented by congenital and acquired disease of the brain. The physician must first differentiate the conditions which are really due to an extensive organic lesion of the brain from such conditions as cretinism and mongolian idiocy in which the mental deficiency is due to other causes. Having done this, the separation of the congenital and acquired cases depends entirely upon whether or not there is a history of a previous disease adequate to produce a widespread organic cerebral lesion. In all cases in which there is no such history, it is best to assume that the mental deficiency is due to a congenital developmental anomaly. When there is a history of pre-existing disease, it must be of such a character as to produce widespread damage to the cerebral tissues. Many acquired organic diseases of the brain are of so local a character that they do not result in defective mentality, but only in some anomaly of motor or sensory function. The disease which most often produces defective mentality and a clinical picture which precisely resembles that seen in congenital defective cerebral development, is meningitis. Acute encephalitis is another possible cause. Hemorrhage most often produces only local manifestations, but occasionally a meningeal hemorrhage is followed by porencephalus and interference with mental development. Hydrocephalus is an obvious cause of mental deficiency of organic origin.

Cretinism.—Cretinism is distinguished from deficient mentality of organic origin by its other characteristic manifestations, such as the shortening of the limbs, and the thickening of the skin and mucous membranes. Furthermore, cretinism is never accompanied by any spasm, paralysis, or signs of cerebral irritation. If any such symptoms are present they may be considered as stigmata of an organic lesion of the brain, and cretinism can be excluded as a cause of the deficient mentality.

Mongolian Idiocy.—The same considerations apply to mongolian idiocy. This condition is recognized by the characteristic appearance of the child and by the absence of any stigmata of organic disease of the brain.

Amaurotic Family Idiocy.—While this condition is characterized by idiocy combined with spastic paralysis of all four extremities, it is distinguished from cerebral defect of developmental origin by the fact that it is invariably characterized by blindness from optic nerve atrophy and by the peculiar cherry-red macula lutea. Furthermore, development is usually normal until the child is eight or ten months of age.

Retarded Mental Development.—We should be very careful in very young children not to confuse slow or retarded development with defective cerebral development. There is so much variation in the time at which children walk and talk, that a delayed development of these functions must not be considered to represent a condition of mental impairment. Some children develop so slowly, both bodily and mentally, that they appear very backward in comparison with others of the same age. Children in the first year of their lives may be so seriously affected by some grave disease that their development is prevented from advancing normally, and in comparison with other children of the same age they may be far below the usual grade of intelligence. If, however, we examine this class of cases carefully, we see that, although they are very backward in their development, they are gradually developing, and that they do not represent the condition of complete arrest of development which exists in mental deficiency from organic disease. Furthermore, all the stigmata of organic disease are absent in simple retarded psychical development. The condition is simply that of a normal child of a younger age, whereas in defective cerebral development, the mental condition of the child is not only merely *backward*, but *abnormal*.

The principal point in differential diagnosis is that the child with retarded development presents no definite abnormality. He is simply backward and his mental condition is that of a normal child of younger age. In mental deficiency from organic disease of the brain, development is not only retarded but soon becomes abnormal. Another most important point in the differential diagnosis is the presence of any stigmata of organic disease of the brain. If there is any paralysis, rigidity, spasm, nystagmus, abnormalities of reflexes, or any other phenomenon characteristic of disease of the motor areas of the brain, simple retarded development can be excluded and the diagnosis must be defective cerebral development. In short, the combination of mental deficiency with any form of congenital cerebral paralysis is positively diagnostic of this disease.

Spastic Paralysis from Other Causes.—Cases in which spastic paralysis is the sole clinical manifestation of congenital cerebral defect must be differentiated from cases in which spastic paralysis is due to other causes. The essential feature in the diagnosis of spastic paralysis from defective cerebral development is its congenital nature. While it may not be apparent immediately after birth, its development in the early months of infancy without any acute condition which could act as a cause, is sufficient evidence of its congenital nature. The only condition with which it is likely to be confounded is the spastic paralysis which sometimes follows cerebral hemorrhage of the newborn. In this condition, in babies who survive the first weeks of life, there is at times an early development of a spastic paralysis. The paralysis which follows cerebral hemorrhage of the newborn is usually of a hemiplegic type, while the paralysis of defective cerebral development is in the majority of cases either paraplegic or diplegic. The two etiological conditions cannot, however, always be differentiated from one another, and there is no way of distinguishing defective cerebral development with spastic paralysis as the sole manifestation from cases in which spastic paralysis is due to intra-uterine hemorrhage. Such a distinction, however, is entirely unnecessary for purposes of prognosis and treatment.

PROGNOSIS.—If the diagnosis of congenital defective development of the brain is made, the prognosis is bad in so far as recovery is concerned. This is obvious from the nature of the lesion. A large number of these cases die during infancy from the various intercurrent affections to which infants are particularly liable. A certain number of cases survive the period of infancy, and in them the prognosis is remarkably good as regards life. Indeed, some cases with defective cerebral development are particularly strong physically, and sometimes even in infancy their nutrition appears to be better than that of the average infant. The outlook as to mental improvement is less good. In one sense there can never be any real improvement, for the underlying condition remains unchanged. On the other hand, the brain normally continues to develop throughout childhood, and even in a brain handicapped by a congenital lesion, a certain amount of development is possible in the unaffected portions. There is consequently in many of these cases a progressive mental development which, however, never reaches the normal. The degree of mental development which can be attained in these cases, and the possibility of the child leading a future life which is at least partially useful, depend not only upon the extent of the lesion, but also upon the skill with which treatment is employed.

The prognosis as to improvement in cerebral paralysis due to a lesion acquired in uterine life is bad. A certain amount of improvement occurs in some cases as the child grows older.

TREATMENT.—The treatment of these cases is entirely by education and training. It is never a matter either for the pediatrist or for the general practitioner, but requires the services of an expert. There are many schools for the feeble-minded in which such education and training is most skilfully carried out. Unfortunately most of these institutions are continuously full, and they are not adequate to care for all the defectives which are found among the poorer classes. The question often arises as to the age at which one of these defectives should be sent to an institution. The difficulty which attends the care of such cases often makes the parents anxious to get rid of the defective child. Most of the institutions for the care of these children are so full that they cannot take patients simply for the purpose of relieving their parents of their care, and they must confine themselves to taking patients who have reached the age at which the beginning of education and training is indicated. It is usually difficult to do much with these children until they have reached the age of six years. Consequently in the majority of cases, parents must be told that it is impossible to get the child into an institution until it has reached that age and that they must do the best they can with its care until then. The results of education and training in the hands of an expert, in the higher grades represented by the imbeciles and morons, are often most surprisingly good.

MONGOLIAN IDIOCY

This is a condition characterized by mental deficiency, and certain peculiar physical characteristics. As the mental deficiency is probably due to defective cerebral development, the condition would be classified as a type under that heading were it not for the fact that the peculiar physical characteristics of the disease are suggestive of an etiology which is entirely different from that which produces the ordinary congenital malformations. Under this view the defective development of the brain is secondary to some other cause.

ETIOLOGY.—The cause of mongolian idiocy is entirely obscure. There is no hereditary factor nor any evidence of the influence of syphilis or alcoholism in the parents. The only factor which has appeared to be of any importance is the age of the mother. In the majority of cases of mongolian idiocy, the mother is over thirty-five years of age, and it is not uncommon in such families for all the children to be normal except the last child, who is a mongolian idiot. At times, however, it is the first child which shows this condition, but it is much more likely to be either the first or the last child than those in the middle. These facts would suggest that the cause of mongolian idiocy is closely connected with the reproductive function. Mongolian idiots have been described by some writers as "exhaustion products."

The condition is not uncommon; indeed it is one of the most common forms of mental deficiency, particularly in England and this country, although this may be due to the fact that it has attracted more attention. In my personal experience, the condition has been more frequently encountered than either defective cerebral development or cretinism.

PATHOLOGICAL ANATOMY.—The brains of mongolian idiots are usually somewhat smaller than normal. The convolutions are poorly developed and the cortex is apt to be thin, with a fewer number of ganglion cells than normal. There are no definite localized lesions.

SYMPTOMS.—The mental development of mongolian idiots is very backward. The condition, however, appears to be a general retardation of development rather than an abnormality of development, such as is seen in cases of defective development. Indeed, were it not for the physical stigmata of the disease, the condition would probably be diagnosed as retarded cerebral development rather than as any form of idiocy. Mongols often do not hold up their heads until they are a year or more old. They may not walk until they have reached the third or fourth year. Speech is greatly retarded, but the majority of cases learn to talk to some extent, although speech is seldom normal. While there is a certain amount of variation in the mental development finally attained, it is always much below the normal, and the majority of these children do not reach a higher degree of mental development than that seen in a normal child of four or five years. These children are restless, inattentive, and can be taught only with the greatest difficulty.

The appearance of the mongolian idiot is so characteristic that it can be seen at once whence this form of idiocy derives its name. The face has a peculiar cast. The three most important signs, of which the first two are always present, are (1) the mongolian slant of the eyes the outer canthus being higher than the inner canthus; (2) the broad flat bridge of the nose; (3) overdevelopment of the epicanthic fold, causing it to cover up the angle of the eye. In addition to these very characteristic signs there are others which are often found, but which are not constantly present. The palpebral fissures of the eyes are usually narrow. The head is usually one or two inches below the normal in circumference. The muscles are poorly developed and the ligaments show a high degree of relaxation, so that the limbs may easily be placed in all sorts of strange and uncomfortable positions. The tongue may be prominent and protrude, as in the cretin. There may be drooling from the mouth or a nasal discharge, and mouth-breathing is frequently present. The nasopharynx is small, and consequently a very moderate amount of adenoid growth will often give the signs of nasal obstruction. For this reason, the appearance of the mongolian idiot is often attributed simply to adenoids, with mental dulness. There are often associated congenital malformations, particularly congenital disease of the heart. In the last six mongolian idiots which have come to autopsy at the Infant's Hospital, congenital cardiac lesions were found in four.

DIAGNOSIS.—The diagnosis of mongolian idiocy depends upon the peculiar physical characteristics seen in this disease. When a child has lived to be old enough for defective mental development to become apparent, the mongol appearance of the child usually gives an easy clew to the cause of the condition. The question arises, however, whether mongolian idiocy can be recognized from the appearance of the child in early infancy before defective mental development becomes definitely recognizable. Are we justified in telling parents that their child is going to be an idiot simply because its facies presents the mongol characteristics in early infancy? I do not think it wise to tell parents of the suspicion of mongolian idiocy until they themselves have noted signs of defective mental development, although it may be wise, when the physician himself becomes convinced that he has to do with a case of mongolian idiocy, to tell some member of the family. It must be remembered that infants who are very poorly nourished, or who show a more or less marked degree of infantile atrophy, often have a facies which more or less resembles that seen in mongolian idiocy. In such cases the physician should not make a diagnosis of mongolian idiocy until signs of defective mentality are plainly apparent. When, however, a young infant is normal in every other respect, but presents the mongol appearance in early infancy, the chances are strongly in favor of its being an idiot, and it is well to warn some member of the family of the possibility of this condition.

When defective mentality is plainly apparent, the diagnosis is much easier. Cases of defective mentality from organic disease of the brain do not show the mongol characteristics. The only condition which presents difficulty in differential diagnosis is *cretinism*. Mongols often have the protruding tongue of the cretin, and marked shortening of the fingers. On the other hand, cretins sometimes show a facies which is suggestive of mongolian idiocy. The differential diagnosis depends mainly on the shortening of all the long bones which is present in cretinism but absent in mongolian idiocy, and on the thickening of the skin, which is seen in cretinism. Most cretins do not have the mongolian cast of countenance. Furthermore, the cretin is much more apathetic, and shows much less response to his surroundings and to various stimuli than does the mongol. In doubtful cases the therapeutic test with thyroid extract should be employed.

PROGNOSIS.—The outlook in mongolian idiocy is not only bad as to mental development but also serious as to life. Mongolian idiots show a singular lack of resistance against infection. Infections which in normal children run a very mild course, are often fatal in the mongolian idiot, and these cases are also particularly susceptible to tuberculosis. The majority of cases die as a result of infection, either in infancy or in early childhood. While many cases of mongolian idiocy are seen in infants' hospitals, few cases are encountered which are more than eight or ten years old. The few cases which survive remain mentally defective throughout life.

TREATMENT.—No treatment is of avail in this condition. As compared with deficient mentality from defective cerebral development, little can be done in mongolian idiocy with education and training. No drug or glandular extract has been found to have any influence upon the condition.

II. MECHANICAL INJURIES FROM EXTERNAL OR INTERNAL CAUSES

TRAUMATIC INJURIES

The majority of injuries to the nervous system from trauma are of surgical rather than medical interest. The effect of a traumatic injury upon the head may be fracture of the skull with the resulting intracranial hemorrhage and injury to the nervous tissue, or the shock of the injury may produce intracranial hemorrhage without fracture of the bones. Under such circumstances the symptoms are essentially those of intracranial hemorrhage, which will be described below.

Traumatic injury to the spinal cord takes place usually as a result of fracture or dislocation of the spine. It is unnecessary to describe in detail the clinical manifestations of such a lesion, which show no characteristics peculiar to early life. The distribution of the paralysis and disturbance of sensation which result from an injury to the spinal cord varies with the situation of the lesion. Traumatic injury usually involves the cord as a whole, rather than any one of its particular tracts, and for this reason the symptoms are both paralysis and absence of sensation. In any localized lesion involving the entire thickness of the cord to a greater or less degree of severity, the paralysis is of the flaccid type in the muscles supplied by the peripheral nerves arising from the injured portion of the cord, and of the spastic type in the muscles supplied by the peripheral nerves arising from the uninjured portion of the cord below the site of the lesion. The disturbance of sensation bears an obvious anatomical relationship to the situation of the injury. The diagnosis of these traumatic lesions of the cord is obvious from the nature of the injury, and the existence of paralysis and disturbance of sensation. The prognosis is always grave in a traumatic injury. Cases of broken neck or broken back with an injury to the upper part of the cord are usually fatal. When the injury is lower down the prognosis varies with the severity of the lesion. Severe cases with complete paralysis below the point of injury may live a long time in a helpless condition with absence of sensation in the lower extremities, and incontinence of urine and feces. Eventually, in spite of care, bed sores occur, and death is likely to take place as a result of general sepsis. The treatment of these injuries is wholly surgical.

Traumatic injuries to the body and extremities may involve the peripheral nerves. Rupture of the nerves, or compression of the nerves in the callus, often takes place in fractures, with resulting disturbance of function. The nerves may be similarly injured in wounds. The prognosis in these cases, while it varies somewhat with the nature and extent of the lesion, is in general good. Regeneration requires considerable time, usually at least six months. In some cases function is not restored.

One condition of external traumatic injury to the nervous system is treated medically, and is frequently encountered in children by the general practitioner. This condition is *concussion.*

CONCUSSION

ETIOLOGY.—This condition is due to a fall or blow upon the head which is sufficient to produce a temporary disturbance of function in the central nervous system, but which is insufficient to produce grave and permanent lesions like those described above. It is not uncommon in older children, the injury usually occurring as a result of their activities in play.

PATHOLOGICAL ANATOMY.—It is probable that the injury produces slight or transitory lesions, although as the condition is not fatal little is known as to the anatomical cause of the symptoms. There may be only a brief alteration in the cerebral circulation, giving rise to very transitory symptoms, or there may be cerebral edema, minute hemorrhages, or slight laceration of the brain tissue.

SYMPTOMS.—The usual history is that of a hard bump upon the head, either from a fall or from a blow. This is usually followed either by an alteration of consciousness with dizziness and sometimes with hallucinations of sight and hearing, or by complete loss of consciousness. This condition of disturbed consciousness is usually only transitory, lasting from a few minutes to perhaps half an hour. After recovery of consciousness the child usually complains of severe headache and vomits a number of times. Sometimes vomiting is obstinate and severe. There is usually no fever; the pulse is most often rapid, but in severe cases may be slow, this probably being due to pressure from cerebral edema. There may be temporary amnesia and aphasia. The symptoms vary in duration according to the severity of the injury, but in most cases the child is fully convalescent by the expiration of twenty-four hours. If symptoms persist longer than this, a more serious injury should be suspected, and a careful examination should be made for paralysis and for other evidences of disturbance of the function of the central nervous system. In some cases epilepsy has developed at some time after such an injury, this being evidence that the injury produced some permanent organic lesion.

DIAGNOSIS.—During the stage of unconsciousness it is impossible to know whether the case is one of simple concussion or whether there is some serious injury, such as hemorrhage or extensive laceration of the brain tissue. A careful examination should be made for evidences of fracture of the skull. The diagnosis of concussion depends mainly upon the absence of complete loss of consciousness, or upon the rapid recovery of consciousness which is seen in these cases. During the subsequent period the physician must have in mind the possibility of a more serious injury and should make a most careful examination for every evidence of disturbance of nervous function. The existence of paralysis, of prolonged unconsciousness, of disturbance of respiration, or of any signs pointing to a localized lesion of the brain suggests that the injury is more serious than simple concussion.

PROGNOSIS.—If the case is one of concussion only, the prognosis is good. Recovery is usually complete as to the function of the nervous system, although in a few cases epilepsy has been known to develop later.

TREATMENT.—The essential of treatment in these cases is perfect rest and quiet in a darkened room. Hot applications may be made to the feet and abdomen, and an ice-bag should be applied to the head. If vomiting is severe or persistent, sodium bromide may be given by rectum in doses proportioned to the age of the child. If the pulse becomes very weak and rapid, the ordinary measures of stimulation may be employed. Treatment should be continued until all symptoms have disappeared.

INSOLATION

(Heat Stroke)

Heat-insolation, or heat-stroke, is a condition apparently represented by a functional disturbance connected with the cerebral circulation and produced by heat. This affection in varying degrees of severity is of somewhat frequent occurrence in children, and is supposed to be accompanied by a hyperemia of the meningeal blood vessels of greater or less intensity, with general venous congestion throughout the body. It is met with most commonly in the middle period of childhood, because at that age the child is most likely to be exposed to the influences which produce it.

SYMPTOMS.—The clinical picture of this class of cases is, as a rule, quite characteristic. The child has perhaps been playing on a hot summer's day somewhat more vigorously than usual, possibly romping with an older child of more highly developed nervous resistance, getting intensely excited, and greatly overtaxing its muscular strength. It may be that it has been exposed to the direct rays of the mid-day sun, or it may have been playing in some covered but heated and stifling place. The child's nurse, noticing the extremely flushed condition of its face and head and its excited, sparkling eyes, takes alarm and hurries it to its home. Intense headache soon comes on, and in a few hours delirium may supervene. The skin is hot, dry, and reddened; there may be vomiting in the beginning; the carotids and temporal arteries throb perceptibly. The heart's action is violent, and the temperature is raised to 102°-103°-104° F.; the pulse is much accelerated, perhaps 140 to 150, and is full, but usually rhythmical. The conjunctivae are congested and the pupils contracted. Photophobia to a greater or less degree is almost invariably present. Beyond this there may be no symptoms except a slight amount of muscular twitching, and in some cases a convulsion may occur if the temperature runs as high as 104° to 105° F. The temperature, however, in accordance with the rule in this disease as in others which occur in children, does not always produce the same or equally severe symptoms. Convulsions may occur as a very common form of nervous explosion when fever and disturbance of the cerebral circulation are present, but, as a rule, this symptom is absent.

PROGNOSIS.—We should be careful as to the prognosis given in these cases. Although they often simulate closely a beginning meningitis, yet they are very amenable to treatment, and should therefore be carefully differentiated from that disease. In very severe cases the children may, of course, die of insolation.

DIAGNOSIS.—The diagnosis from meningitis is based upon the history, the milder grade of the symptoms, except the headache, and finally, in doubtful cases, on the rapid recovery and speedy disappearance of the fever.

TREATMENT.—The treatment of heat-insolation with severe symptoms and high temperatures should be prompt and vigorous. A stimulating enema of salt solution, one teaspoonful to a quart of cold water, should first be given. The child should then be placed upon a bed protected by a rubber sheet in a cool, darkened room. A warm mustard pack should be applied to the lower extremities, and the neck and chest sponged with water at 77° F. for fifteen minutes out of every hour. Leiter's coil should be applied to the head with water at 41° F.; bromide of sodium should be given, 5 grains every hour for four doses; a little iced milk may be taken if the child cares for it, not more than one or two ounces at a time; and complete rest and quiet for at least twenty-four hours are usually indicated. Stimulants by mouth and subcutaneous injection should be given only if needed for severe collapse. The child should be watched carefully for some days and not allowed to play actively enough to get heated. Great care should be taken for the rest of the summer to protect the child from the direct rays of the sun, as after one attack the cerebral circulation remains in a very sensitive condition for a considerable period.

Mild cases occur which do not call for such vigorous treatment. Rest in bed, small doses of brandy or aromatic spirits of ammonia, and a light diet of milk and lime-water are all that is required in these cases.

CEREBRAL HEMORRHAGE

Intracranial hemorrhage is seen most often in the newborn, either as a result of some injury during intra-uterine life, or more commonly as a result of some injury received during parturition. Cerebral hemorrhage from these causes plays such an important part in the diagnosis of the diseases of the newborn, that it has been described in that division. Sometimes, however, the symptoms are not encountered until the child is very much older, and in such cases it is difficult to know whether the hemorrhage took place in the newborn or at a subsequent period.

Cerebral hemorrhage may also take place from various causes acting during extra-uterine life. In the majority of text-books this condition is described under the heading of cerebral paralysis, the latter term representing a symptom complex which is due to several different causes and lesions. The two principal causes of cerebral paralysis as thus described are cerebral hemorrhage and acute encephalitis. Other less common causes are cerebral thrombosis and cerebral embolism.

ETIOLOGY.—The most common immediate cause of cerebral hemorrhage developing in extra-uterine life is *trauma*. Nevertheless in many cases there is no definite history of a traumatic injury, the trauma having been too slight to be noticed. Furthermore, cerebral hemorrhage is very rarely seen after the first five years of life. Hemorrhage is, however, seen comparatively often in the first five years of life, although at this age the conditions leading to traumatic injury are not encountered so frequently as in older children. The comparative frequency of occurrence of hemorrhage at this age can only be accounted for by the presence of some other factor connected with the age of the patient, which so increases the tendency toward rupture of the blood vessels that a comparatively slight traumatic injury is sufficient to produce a hemorrhage. This can only be the increased delicacy and susceptibility to shock of the meningeal vessels in infancy and early childhood. It is quite probable that this delicacy of the blood vessels may be increased by the various forms of disease peculiar to this period of life, although in many cases the connection is not very close. Besides the association of comparatively slight trauma with delicate meningeal vessels, a certain number of other causes have been connected with the occurrence of meningeal hemorrhage. Among these are pachymeningitis, and thrombosis of the superior longitudinal sinus. Cerebral hemorrhage has been seen as the result of the acute hyperemia produced by a paroxysm of pertussis.

PATHOLOGICAL ANATOMY.—In the great majority of cases the hemorrhage is meningeal, the blood usually being poured out into the subarachnoid space. This is the most important difference between the cerebral hemorrhage of early life and of adult life.

It should always be remembered that when hemiplegia in a child can be traced to cerebral hemorrhage, the hemorrhage is not into the internal capsule, as in the adult, but is almost invariably into the subarachnoid space over the convexity of the brain. This difference is due to the fact that arteriosclerosis, the most common cause of cerebral hemorrhage in adults, is not seen in early life. The pathological anatomy of these meningeal hemorrhages is the same as was described in cerebral hemorrhage of the newborn. There is usually a more or less extensive pouring out of blood which forms a clot of variable size over one hemisphere of the brain. As this hemorrhage usually involves more or less of the cortical motor centers, the result is paralysis. Rare cases are seen in which the hemorrhage takes place into one of the ventricles or into some other part of the brain, but this is exceedingly uncommon in the acquired form.

The later results of hemorrhage depend upon its extent and upon how great is the pressure injury to the cells of the cerebral cortex. In the mildest cases the extravasated blood is entirely absorbed without leaving behind any permanent injury. In other cases, fibrous tissue organization takes place in a certain portion of the clot, and this may result in a more or less constant pressure upon and irritation of the motor centers of the cortex. In still more severe cases, the brain tissue is so injured that there is atrophy of the motor cells of the cortex, this being followed by a descending degeneration of the pyramidal tracts. From these facts it may be seen that an old hemorrhage may be followed either by no symptoms, by symptoms of cortical irritation without permanent paralysis, or by permanent paralysis.

SYMPTOMS.—The symptoms of cerebral hemorrhage may be divided into those which immediately accompany the hemorrhage and those which develop later. In some cases the early symptoms are unnoticed either because they occur at night when the child is not under observation, or because they are too slight to attract attention. In the majority of cases the onset is sudden, the most characteristic symptom being convulsions. The convulsions in cerebral hemorrhage usually differ from those characteristic of spasmophilia and of reflex and toxic causes in that instead of lasting only a few minutes, they are likely to be severe and prolonged. They often persist for half an hour or an hour, and sometimes the child goes into an eclamptic state which lasts for many hours, with only slight remissions. The convulsions may be general. In some cases, however, there is a tendency toward an irregular distribution of the convulsive movements, one side of the body being involved more than the other, or the face or one limb may be involved more than other parts of the body. This irregularity of distribution is a very important diagnostic sign, when present.

Consciousness is usually completely lost during the convulsions. Between convulsions consciousness may return, or the child may remain unconscious. In severe cases, loss of consciousness may persist even after the convulsions have ceased. In mild cases, the convulsions are of brief duration and the child soon returns to consciousness. In addition to convulsions and disturbance of consciousness, there may be various signs of irritation of the motor areas in the cortex, such as twitching of the limbs and dilatation or contraction of the pupils. Fever is usually entirely absent, this being one of the most important signs in the differentiation from acute encephalitis. In some cases the attack is ushered in by screaming or delirium. Vomiting is not uncommon. It should never be forgotten, however, that the symptoms may be so mild as entirely to escape attention.

The paralysis may become evident before these early symptoms have subsided, or may follow them closely. The most common type is *hemiplegia*. This is usually accompanied by a unilateral facial paralysis, although in some cases only the arm and leg are affected. When facial paralysis occurs, the upper segment of the facial nerve is not involved, so that the eyes can be closed and the brows raised. This is a sure sign that the facial paralysis is of central and not of peripheral origin. In some cases there is monoplegia. Sometimes the arm and face are affected, or else there is marked paralysis of the leg, with very little paralysis of the arm. There are all sorts of variations in the extent of the paralysis, but the essential point is, that in acquired cerebral hemorrhage, the type is almost invariably hemiplegic. Diplegia and paraplegia are only seen in the cerebral hemorrhage of the newborn.

On examining the paralyzed limb we find a resistance to passive motion, the deep reflexes are exaggerated, and in most cases there is a feeling of rigidity on the paralyzed side. Slight ankle clonus is often present. Sensation, as a rule, is not affected. When the child has come out of its stupor and the convulsions have ceased, it may be found to be aphasic, but aphasia is not so common as in the cerebral lesions of adults.

The paralysis never remains as extensive as it is when it first develops. After a period which may vary from one to two weeks, the patient begins to show an increasing use of the paralyzed limbs, usually first the leg and then the arm. Facial paralysis, if present, passes off still earlier. Aphasia, which is usually seen with right hemiplegia, may sometimes in childhood be seen when the paralysis is on the left side. It almost invariably disappears within a few days. In infants who have not learned to walk, this function is acquired late, and there may be various peculiarities of gait, usually a dragging of one leg. In older children who have been able to walk before the attack, the power of walking is ordinarily regained after a few weeks, although there is usually dragging of the affected leg. Recovery in the leg is often complete, although a slight halt in the gait may persist permanently. The arm usually recovers more slowly than the leg, and in many cases a certain amount of paralysis becomes permanent. In still other cases, recovery is complete both in the arm and in the leg. The time which elapses before restoration is complete, or the limit of improvement has been reached, is very variable.

In the cases in which paralysis persists, contractures are likely to develop. These produce various deformities. In the lower extremities contractures lead to the various forms of talipes. In old cases the paralyzed limbs show a varying amount of atrophy, and there is more or less rigidity, which is sometimes quite marked. Athetosis is rarely seen as a sequela of cerebral paralysis from hemorrhage.

In the majority of these cases of acquired cerebral hemorrhage, the mental condition is wholly normal, this being in marked contrast with the cases in which cerebral paralysis is due to defective development of the brain, or to cerebral hemorrhage of the newborn. If, however, the hemorrhage occurs in very early infancy, it may interfere sufficiently with the development of the brain to produce impairment of the mentality. Epilepsy is not uncommon as a sequela of cerebral hemorrhage. In some cases the convulsions are general, but more often the epilepsy is of the Jacksonian type. The sphincters, of course, are never affected in cerebral paralysis.

DIAGNOSIS.—The diagnostic problem which confronts the physician in these cases varies according to whether he sees the case during the period of onset before definite paralysis has developed, during the period of paralysis which follows the onset, or at a later period after the limit of improvement has been reached. In the period of onset he is confronted by a condition characterized mainly by convulsions and more or less disturbance of consciousness. He must take into consideration all the causes which may produce such a condition. The convulsions which are due to spasmophilia or reflex disturbance are almost always of briefer duration or not so frequently repeated, and do not produce a notable disturbance of the consciousness except during the actual occurrence of the convulsion. The convulsions which in children sometimes usher in an acute infection, are not usually severe or prolonged, and can be excluded by the absence of a marked febrile reaction. When convulsions are prolonged and repeated, it is a fairly safe conclusion that they are due not to a secondary disturbance of function but rather to some acute organic disease of the brain. Only acute diseases need be considered, and among them there are three important possibilities. These are (1) hemorrhage; (2) acute encephalitis; (3) acute meningitis. If there is fever, while hemorrhage cannot be positively excluded, the probabilities are in favor of the lesion being either acute encephalitis or acute meningitis. The differentiation between these two conditions will be discussed later. In this stage the physician is only justified in making a probable diagnosis of cerebral hemorrhage, if the condition which presents itself is one of prolonged convulsions with disturbance of consciousness and without fever. A history of any trauma which might account for hemorrhage makes such a diagnosis fairly positive.

In a later stage of the disease the physician is confronted by the picture of a cerebral paralysis with a history of an acute onset. The only form of *meningitis* which may have no fever at the onset is the tuberculous. It is only in rare cases that a tuberculous meningitis begins with convulsions, and still more uncommon that any form of meningitis shows so early a development of paralysis of a hemiplegic type. Consequently meningitis can usually be easily excluded in this stage of the disease. The only other condition which is likely to cause a suddenly developing cerebral paralysis is an *acute encephalitis*. In the majority of cases an acute encephalitis represents simply one of the manifestations of poliomyeloencephalitis. The differential diagnosis of this disease has already been discussed in detail. The most important points are the febrile onset, and the fact that the cerebral paralysis seen in the encephalitic form of infantile paralysis is rarely of a definitely hemiplegic type, but is usually monoplegic, diplegic or paraplegic. Cases occur, however, in which an acute encephalitis not due to the microörganism which produces poliomyeloencephalitis is encountered in early life. Such cases usually appear as complications of one of the recognized acute infections. The paralysis is quite as likely to be hemiplegic as it is in cerebral hemorrhage. The principal differentiating point is the distinct febrile reaction which always accompanies the acute stage of any form of encephalitis, but which is usually entirely absent in cases of hemorrhage. It should be remembered that acute encephalitis is a more common cause of cerebral paralysis than is hemorrhage, and unless a distinct history of trauma is present, a diagnosis of hemorrhage can only be made when there is a complete absence of fever during the onset of the disease.

When the patient is first seen at a still later stage, an etiological diagnosis is extremely difficult. In this stage the physician is confronted by a certain amount of paralysis and rigidity which may be confined to a single limb, or may be of hemiplegic distribution. The rigidity, the increased deep tendon reflexes, the comparatively slight wasting, the normal electrical reactions and the distribution of the paralysis, usually enable one easily to locate the lesion in the cortex of the brain. Consequently it is easy enough to make a diagnosis of old cerebral paralysis. Such a diagnosis is all the more strengthened if Jacksonian epilepsy, or choreic or athetoid unilateral movements be present. It is much more difficult, however, to determine the etiology of the condition. The diagnosis depends entirely upon how accurate a history of the acute attack can be obtained. The considerations which are used in making a diagnosis are the same as those which enter into the differential diagnosis of the acute stage of the disease. Many cases are encountered, however, in which all that the physician can find is the slight dragging of one leg, possibly combined with epilepsy. In these cases he can only say that the condition is due to an old cerebral lesion, which may have been either hemorrhage or encephalitis.

PROGNOSIS.—Cerebral hemorrhage occurring in extra-uterine life is very much less frequently fatal than is either the cerebral hemorrhage of the newborn or the cerebral hemorrhage of adults. In cases in which the hemorrhage is due to slight or unnoticed trauma, to a paroxysm of pertussis, or to pachymeningitis or thrombosis, death practically never occurs as a result of hemorrhage. A fatal ending is only seen in cases caused by a traumatic injury so severe as to produce fracture of the skull or extensive laceration of the cerebral tissues. As to the paralysis, there is wide variation in the outcome. In individual cases there is always marked improvement. The extent of the improvement finally attained depends more upon how rapidly it begins and progresses than upon the extent and distribution of the paralysis. Facial paralysis usually disappears entirely. In hemiplegia, entire recovery may occur, but usually there is left at least a slight limp or dragging of a leg. In other cases, more or less permanent paralysis remains in the arm, leg or both, with eventual contraction and deformity. The liability to the subsequent development of epilepsy must always be remembered, and this sequela is just as likely to be encountered in mild cases which show almost complete recovery as in the more severe ones. In general, we can say that if after a few weeks there is marked improvement in the child's ability to use the paralyzed limbs, the prognosis is fairly good; if on the contrary there is no improvement for months, it is bad.

TREATMENT.—During the acute stage characterized by convulsions or other signs of cortical irritation, the child should be kept absolutely quiet in bed, with an ice-bag or Leiter's coil applied to the head, and hot applications to the feet and abdomen. Bromide should be freely given in doses proportioned to the age of the child; if the bromide be vomited, it should be given by rectum. If the convulsions are prolonged and severe, and resist the administration of bromide, inhalations of ether should be given from time to time, according to the severity of the convulsions. If the child tends to go into convulsions as soon as it comes out of ether, a dose of chloral may be given in addition to the bromide. The doses of sodium bromide and chloral appropriate to the different ages have been given in Volume I, page 170.

During the stage of improvement after the acute symptoms have subsided, the child should at first be kept quiet, and then gradually allowed to resume whatever activity is possible. Massage is useful in this stage, but electricity has not given sufficient evidence of value to warrant its use. The physician should make sure that paralyzed limbs are kept in a normal position to prevent the occurrence of early contracture and deformity.

After the limit of improvement has been reached, the treatment is orthopedic, with massage, training, apparatus, and sometimes tenotomy.

CEREBRAL THROMBOSIS

The most common form of cerebral thrombosis is due to infection, and this condition will be described under the infectious diseases of the nervous system. There is a form of thrombosis usually described as cachectic or marantic, which is not due to infection.

ETIOLOGY.—This form of thrombosis is usually secondary, occurring in the course of some disease which has produced an extreme reduction of the child's vitality. It is seen at times in conditions of infantile atrophy, extreme anemia or exhausting diarrhea, and sometimes occurs in the course of such acute infections as pneumonia, pertussis, diphtheria, nephritis and tuberculosis. It is not common. Cerebral symptoms often occur in the course of the above mentioned infections, and are sometimes wrongly attributed to thrombosis; in the majority of cases they are not due to cerebral thrombosis, but to irritation of the cerebral tissues or the meninges by the toxins of the disease. The actual cause when thrombosis occurs is the condition of lowered vitality, leading to feebleness of the circulation and an altered condition of the blood. The lesion is seen more often in infancy and early childhood than in later childhood and adult life.

PATHOLOGICAL ANATOMY.—It is the sinuses of the dura mater which are usually involved in cachectic thrombosis. The most frequent seat of the lesion is the superior longitudinal sinus. Clots are formed which fill the sinus completely, are adherent, and sometimes show a laminated structure. The thrombosis usually extends from the sinus into the veins emptying into it. The condition may be complicated by hemorrhage, or the brain and meninges may simply show edema.

SYMPTOMS.—The symptoms of cachectic thrombosis unaccompanied by hemorrhage are few and uncertain in the majority of cases in which this lesion is found post-mortem. The disease is often wholly latent. Even when symptoms are present, they are not often sufficiently characteristic to permit a diagnosis during life. The symptoms are those of meningeal or cortical irritation, and are indistinguishable from symptoms produced by much more common conditions.

PROGNOSIS.—The prognosis is bad. The majority of cases die in the course of a few days.

TREATMENT.—In view of the impossibility of a definite diagnosis, the treatment must be wholly symptomatic.

CEREBRAL EMBOLISM

This condition is very rare in early life. It is practically only seen as a complication of ulcerative endocarditis. The symptoms are much like those of cerebral hemorrhage. The diagnosis depends upon the association of the symptoms of cerebral hemorrhage with ulcerative endocarditis. The treatment is the same as that of cerebral hemorrhage.

FACIAL PARALYSIS

This name applies to a clinical condition due to a variety of causes which may be central or peripheral.

ETIOLOGY.—Central facial paralysis is a not uncommon symptom of *poliomyeloencephalitis*. In this disease it is usually but not always associated with paralyses in other situations. Central facial paralysis may also be part of the paralysis which occurs as a result of *cerebral hemorrhage*, or from a secondary *acute encephalitis*. In these conditions it is usually but not always accompanied by hemiplegia. The principal diagnostic point in the differentiation of a central from a peripheral facial paralysis is that in the former only the two lower segments of the nerve are involved, the patient being able to close the eye and to wrinkle the brow, while in peripheral facial paralysis all three segments are involved. It is only the peripheral form which is due to a traumatic or mechanical lesion, and therefore only this form will be considered here.

The form of facial paralysis caused by an injury to the facial nerve during *parturition* has been described under Diseases of the Newborn. In extra-uterine life the facial nerve may be injured in any one of three situations, within the cranium, in the bony canal, and after its exit from the cranium. Mechanical injury to the nerve within the cranium may be due to the pressure of a *cerebral tumor*, to trauma in *fracture of the skull*, and to involvement in the exudate of a *basilar meningitis*.

In the bony canal the injury to the facial nerve usually comes as a result of disease of the ear. It is rarely seen with acute otitis, but usually occurs as a result of a *chronic otitis* with caries of the petrous portion of the temporal bone. The actual injury to the nerve is mechanical from pressure of the inflammatory exudate.

The commonest form of peripheral facial paralysis is that due to an injury to the nerve after its exit from the stylomastoid foramen. The usual cause is *exposure to cold*, which produces an inflammatory exudate in the sheath of the nerve. In rare cases the mechanical injury comes from the enlarged parotid of mumps or from an enlarged lymphnode.

PATHOLOGICAL ANATOMY.—In the majority of cases the injury is not sufficient to cause degeneration of the nerve fibres, the functional disability being caused by pressure either of the exudate within the sheath of the nerve, or of something outside the nerve sheath. In some cases, however, the injury is sufficient to produce degeneration of the nerve fibers. This may even take place in the form caused by exposure to cold.

SYMPTOMS.—The most typical form is that due to exposure to cold. There is often a history of the patient being exposed to a severe draught of cold air, or sleeping in a draught. I have seen the condition most often occur when the child has slept with its head near the crack of a window opened at the bottom. The paralysis appears suddenly without any other symptoms. It involves the muscles of the forehead, cheek, nose, mouth, and those about the eye. There is an absence or diminution of the normal naso-labial fold on the affected side, which is smooth, and the child is unable to wrinkle the forehead, to contract the eyebrows, to close the eye completely, to whistle, to blow, or to wrinkle up the side of the face. The mouth is drawn toward the unaffected side, and the naso-labial fold on that side is deeper than normal. When the paralysis is only partial, it may not be noticeable while the face is at rest, but comes out clearly when the child cries or attempts to alter the expression of its face. Sensation is wholly unaffected. The electrical reactions are the same as those seen in other forms of neuritis, but vary with the severity of the case. In the milder cases in which the symptoms are due only to pressure without degeneration of the nerve fibers, there is only slightly diminished response to the Faradic current, and the reaction of degeneration is absent. In severe cases with injury to the nerve fibers, the reaction of degeneration is present.

In peripheral facial paralysis due to other causes, the physical signs are the same as in the form due to exposure to cold, the other symptoms being those of the underlying disease to which the lesion of the facial nerve is secondary.

DIAGNOSIS.—Facial paralysis is easily recognized by inspection. Its peripheral character is recognized by involvement of the upper segment of the nerve. The diagnosis of its cause depends upon the history of the case. If there is no evidence either of otitis or of intracranial disease, the physician should assume that a peripheral facial paralysis is due to exposure to cold.

PROGNOSIS.—This depends upon the cause. In the form due to exposure to cold, the majority of cases recover completely within a few weeks. Occasionally, however, a case of this type is encountered in which complete recovery does not occur, or only occurs after many months of treatment. Such exceptionally severe cases can be recognized by the presence of a more or less marked reaction of degeneration. The prognosis in facial paralysis due to otitis media is somewhat less favorable. While many cases recover completely, in others some paralysis is permanent. When the cause is some intracranial lesion, the prognosis is that of the underlying disease.

TREATMENT.—In general this is the same as that of the other forms of neuritis, but in facial paralysis the absence of pain and of acute symptoms permits treatment with electricity to be begun earlier than in multiple neuritis. *Faradism* should be used. Strychnin is much used in these cases, but it is very doubtful whether it has any specific influence. Mild cases will recover spontaneously without treatment, but it is best to use electricity in every case which shows a reaction of degeneration.

COMPRESSION MYELITIS

Pott's Paraplegia, Pressure-Paralysis of the Spinal Cord)

This condition is the only other common form of spinal paralysis occurring in early life besides the paralysis of poliomyeloencephalitis.

ETIOLOGY.—By far the most common cause is tuberculosis of the vertebrae. The disease is seen most often when caries of the spine is in the upper half of the spinal column, and according to Gibney, paraplegia occurs in 50 per cent. of such cases. In rare cases compression myelitis may be of traumatic origin. Other rare causes in childhood are tumors of the spinal cord, or a chronic pachymeningitis.

PATHOLOGICAL ANATOMY.—In tuberculosis of the spine there is a softening of the bodies of the vertebrae which produces an angular deformity called kyphosis. The spinal canal is encroached upon not only by the remains of the vertebral bodies, but also by inflammatory products. This results in a gradual compression of the cord, although in some cases there may be sudden pressure from the slipping backward of one of the loosened vertebral bodies.

Macroscopically the cord is slightly or considerably smaller than normal at the point of pressure, according to the severity and duration of the process. Paraplegia may even exist when no macroscopic changes are visible in the cord. The area involved is usually from half an inch to two inches in length. Microscopically in the milder cases no definite lesions are visible. In more severe cases the microscopic lesions are those of interstitial myelitis. In cases which have existed for a considerable time, there is degeneration of the nerve fibers and sometimes more or less disappearance of the ganglion cells. In the most marked cases the inflammatory and degenerative changes reach such an extent that all distinction between the gray matter and the white matter is lost. In these marked cases there is more or less ascending degeneration of the sensory tracts, and descending degeneration of the motor tracts, as in other focal lesions. There is also usually an inflammatory reaction about the nerve roots in the compressed area.

SYMPTOMS.—The principal symptoms of compression myelitis are pain, paralysis and disturbance of sensation. Pain is usually the earliest symptom, and is caused by the inflammation of the sensory nerve roots. The pains are usually acute and more or less paroxysmal, and are not referred to the spine but to the region corresponding to the distribution of the sensory nerves affected, according to the location of the lesion. These pains are felt in the neck, chest, epigastrium and loins.

The paralysis develops gradually. The most typical form is paraplegia, which comes on with a gradual progressive weakness of the lower extremities. Occasionally, when the lesion is high up, the arms also are affected. The degree of paralysis produced varies mainly with the treatment, but in neglected cases paralysis continues to develop until there is complete paraplegia. This is the commonest distribution, but if the lesion is in the cervical region the arms are sometimes affected as well as the legs. The paralysis in the arms may be of a flaccid type, but that in the legs is spastic causing rigidity of the extremities, a marked exaggeration of all the reflexes, and marked ankle clonus. In the rare cases in which paralysis accompanies a lesion of the lumbar enlargement of the cord, there may be a flaccid paralysis with loss of reflexes, paralysis of the sphincters, and bed sores. Muscular atrophy and contractures follow the paralysis to an extent which varies with that of the secondary lesions in the cord.

Other disturbances of sensation beside the pain are anesthesia, numbness, tingling, or formication. When the disease is in the upper cervical region, death may occur from sudden pressure upon the cord, or there may be vomiting, irregularities of the pupils, hiccough, or occasionally paralysis of the diaphragm.

DIAGNOSIS.—This is rarely difficult. The nature and distribution of the disturbance of function easily enables the physician to diagnose a transverse lesion of the cord. Whenever any child shows the symptom complex of a transverse myelitis, particularly if the symptoms come on slowly, spinal caries should be suspected. Physical examination will almost invariably reveal the deformity of the vertebral column. In the rare cases in which the symptoms of transverse myelitis are due to tumor, the diagnosis is more difficult and can only be made by the exclusion of other possible causes. The cause is obvious in traumatic cases.

PROGNOSIS.—This depends mainly upon the treatment of the case. When the condition is recognized early and proper mechanical treatment applied, the paralysis often passes off in the course of a very few weeks. In neglected cases, the outlook is not so good. The prognosis is also more unfavorable in cases in which paralysis develops while proper mechanical treatment is being carried out. Of course, the prognosis of the underlying tuberculous process must also be taken into account.

TREATMENT.—This should always be a matter for the orthopedist. The indications are the removal of pressure by means of properly adjusted mechanical apparatus. The nursing of these patients is very important, scrupulous cleanliness and frequent changes of position being necessary in order to avoid bed sores. Electrical treatment is contraindicated. Some writers advocate the administration of iodid of potash, given in large doses well diluted. There is no positive proof, however, that this has a favorable influence upon the disease.

III. NEW GROWTHS

New growths of the central nervous system may involve either the brain or the spinal cord. New growths of the brain are much the commoner in early life.

INTRACRANIAL TUMORS

The commonest lesion in infancy and childhood which produces the symptom complex usually associated with intracranial tumor is *tubercle*. Upon a strict basis of classification, however, the large tuberculous masses found in the cerebral tissue are not new growths and cannot properly be considered under this heading. These tuberculous tumors are manifestations of infection, and under an etiological basis of classification they must be considered under the group of conditions due to infection. That they are usually classified among the new growths is due to the fact that they produce functional disturbances of the nervous system, which in every way resemble those produced by true new growths, and they are consequently described with the other tumors in order to avoid repeating the clinical description. In this book, however, the pathological anatomy, diagnosis, prognosis and treatment of the solitary and multiple tubercles of the brain will be considered under the heading of the infections, and only the true new growths will be considered here.

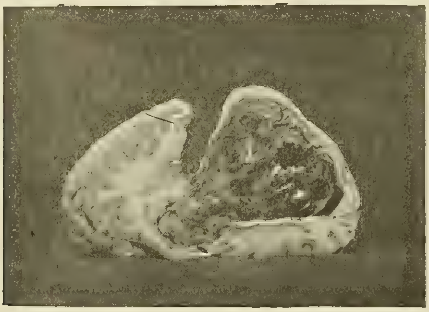
ETIOLOGY.—The cause of cerebral tumors, as of new growths in general, is one of the unsolved problems in medicine. In very few instances is there any history of trauma, or of any other factor which could be considered a predisposing cause. Cerebral tumors may be primary or secondary. In most cases of cerebral tumor in childhood, the new growth is primary in the brain.

PATHOLOGICAL ANATOMY.—The most important forms of cerebral tumor encountered in early life are glioma, sarcoma, gliosarcoma and carcinoma. The cystic tumors of parasitic origin which occur quite frequently in other parts of the world, although not in America, are not strictly to be classified among the new growths, although they are apt to give the same symptoms as intracranial tumors. Gummata of the brain also, while very rare in early life, may present the symptom complex of cerebral tumor, although they also do not belong among the new growths.

Glioma is the commonest form of cerebral tumor encountered in early life. While it is seen more often in childhood than in the earliest years, it is not infrequent in infancy. It represents a primary new growth of the brain, the tissue repeating the structure of the neuroglia. It is consequently composed of connective tissue and branching cells. Gliomata are not sharply bounded tumors, but are markedly infiltrating, so that the limits of the new growth are very difficult to determine even upon microscopic examination. Gliomata may be situated in any part of the brain, but the most common situation is the cerebellum, the pons standing next. In fact, a little the commonest of all the cerebral new growths in childhood is glioma of the cerebellum. It must never be forgotten, however, that any part of the intracranial contents may be the site of the tumor.

Sarcoma stands next in frequency, and indeed is almost as often encountered as glioma. Sarcoma of the brain is usually primary, but in rare instances metastatic sarcomata are found in the cerebral tissues. The sarcoma may be of any of the recognized anatomical varieties,—round cell, spindle cell, and so forth. At times a combination of glioma and sarcoma is present in new growths, the lesion in such cases being described as gliosarcoma. The sarcomata of the brain are often less infiltrating than are gliomata, and sometimes are quite sharply bounded by a fairly definite capsule. They grow much more rapidly than do gliomata. The most common situation for sarcoma is the cerebellum, the pons standing next in frequency, then the basal ganglia, and then the cortex. Both gliomata and sarcomata may undergo cystic softening.

FIG. 277



Sarcoma of the brain in a girl of 2½ years

Carcinoma is very much rarer in early life than either of the two forms described above. It is always metastatic and secondary to a primary carcinoma in some other part of the body.

The growth of tumors of the brain usually produces various secondary lesions caused by pressure on the contiguous cerebral tissues. Pressure on the aqueduct of Sylvius or on the fourth ventricle produces an obstruction which prevents the exit of fluid from the internal ventricles, and in this way an internal hydrocephalus may be produced. Hydrocephalus is a very common complication of all cerebral tumors situated in the posterior fossa. Other results of the pressure of growing tumors are injury to the nerve cells and fibers with corresponding disturbance of the functions of the central nervous system. Frequently there is a localized meningitis over the cerebral tumor, and this may be the cause of some of the symptoms of disturbed function. Rarely cerebral hemorrhage may be associated with new growths of the brain.

SYMPTOMS.—The symptoms of cerebral tumor may be divided into two groups. In the first group are the general pressure symptoms, which may be produced by an intracranial tumor in any situation. In the second group are the localizing symptoms which depend upon the interference with the function of the nervous tissue produced by the growth of the tumor. It should be remembered that in cerebral tumor the clinical manifestations are always those of a chronic rather than of an acute disease, an important point in the differential diagnosis from such conditions as hemorrhage and encephalitis.

GENERAL SYMPTOMS.—The most important and constant general symptoms of intracranial tumors are headache, vomiting, convulsions and choked disc. Other important but less constant symptoms are disturbances of the mentality, disturbances of sleep, vertigo and enlargement of the head. The first symptom noted is very variable; sometimes it is headache, sometimes a convulsion, sometimes unaccountable vomiting, sometimes some disturbance of the disposition or mentality.

Headache is probably the most constant of the symptoms of cerebral tumor. It is apt to be severe, but varies very much in intensity, character and position, not only in different cases but from time to time in the same case. It may be constant, or paroxysmal and intermittent. The pain may be referred to any part of the head, and its location has no relation to the situation of the tumor. In the majority of cases the headache is frontal. It may be accompanied by a sensation of tension in the head, or by localized tenderness of the scalp. Infants and very young children do not complain definitely of headache, and the physician can only judge that they are in pain from their expression and actions. A frowning expression and occasional whimpering is the most common manifestation of severe headache in an infant when the pain is constant, crying only occurring when headache is paroxysmal. Older children complain more definitely and constantly of headache in cerebral tumor than in any other condition.

Vomiting is a symptom which stands next in constancy of occurrence to headache. It is, however, not of so much diagnostic significance, because this symptom is associated with so many more common conditions in early life. It is more significant in older children than in infants. The vomiting in cerebral tumor usually occurs in attacks which come at varying intervals without recognizable cause. The attacks are usually a few days apart, but may occur more or less often. It is significant that these attacks of vomiting occur without nausea or other symptoms of indigestion, and that they are entirely independent of the diet. They are very apt to occur very early in the morning. This symptom may appear early in the course of the disease and persist for many months.

Convulsions are almost as constant as vomiting, and are of more diagnostic significance. They almost invariably appear at some time in the course of the disease, and in the majority of cases they are seen in the early stage. The convulsions which are symptoms of all forms of tumor irrespective of localization are general in character. Localized convulsions are sometimes seen in cases in which the tumor is situated near the motor area of the cortex.

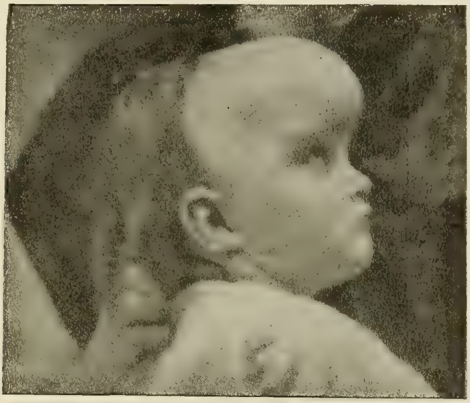
The general convulsions occur at first at quite long intervals, but with the progress of the disease they gradually become more frequent and severe. In the early stages the attacks often resemble petit mal, consisting only of a very brief loss of consciousness with or without slight twitchings. As the disease progresses the attacks become more severe, and all degrees of severity are seen up to typical epileptiform convulsions with complete loss of consciousness. In some cases localized twitchings or convulsions precede the general type. Convulsions as a symptom of brain tumor are most common and severe when the growth is rapid, or when there is an extensive meningitis over the site of the lesion.

Choked disc (optic neuritis or papillo-edema) occurs in about 90 per cent. of cases of intracranial tumor and is probably the most important diagnostic sign. There may or may not be a disturbance of visual function, and the condition can only be recognized with certainty by ophthalmoscopic examination. The lesion is usually double. Choked disc is a constant and early symptom with cerebellar tumors, with tumors of the corpora quadrigemina, and with tumors of the parietal and occipital regions. It is later in appearance and sometimes absent with tumors of the pons, corpus collasum, or convexity.

Disturbance of mentality is usually present at some time in the course of the disease, but is often a later symptom than those mentioned above. The symptoms present the greatest variety. In the early stages there is usually only fretfulness and irritability, or a marked change in the disposition of the child. These symptoms are so common from a great variety of causes in children, that they do not usually attract attention unless accompanied by other manifestations. As the disease progresses there is apt to develop a variable amount of dulness, apathy, or somnolence. In some cases there are attacks of a condition resembling melancholia. In other cases there are periods of excitement which sometimes resemble acute mania. Finally, the mental impairment may become so marked as to suggest a condition of imbecility. The sleep of children with brain tumor is usually markedly disturbed. Insomnia is sometimes marked, and attacks of screaming with various hallucinations are sometimes seen.

Vertigo is a fairly common symptom. In the early stages of the disease it is usually associated either with vomiting or with a sudden change of position. Later it may be constant, especially with tumors situated below the tentorium.

FIG. 278



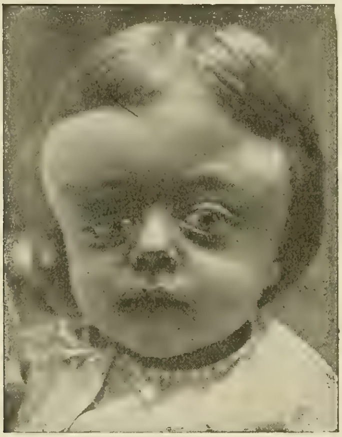
Sarcoma of the brain in a girl of 2½ years

Enlargement of the head is usually associated with the hydrocephalus produced by the tumor. It is seen most often in the first two years of life, before the fontanel has closed and the ossification of the sutures has become complete; but sometimes marked enlargement of the cranium with separation of the sutures is seen in later childhood. Occasionally in young infants a rapidly growing sarcoma produces a localized prominence in some part of the cranium, such as is shown in Fig. 278.

A slow pulse would be expected as a symptom of increased intracranial pressure in cerebral tumor. As a matter of fact, however, slowing of the pulse as a sign of intracranial pressure occurs only when the increase in pressure is either very rapid or very marked. Consequently while slowing of the pulse is sometimes seen with brain tumors, it is by no means a constant symptom and is most apt to appear in the later stages of the disease. The occasional association of diabetes insipidus with brain tumor has been mentioned under the description of that disease.

LOCAL SYMPTOMS.—These appear as the result of pressure upon the brain tissues and depend entirely upon the situation of the tumor, not at all upon its anatomical variety. The pressure may cause only irritation of the nervous tissues, or there may be destruction with paralytic symptoms. It should be remembered that in any case of brain tumor, local symptoms may be entirely lacking. They depend to a great extent upon the rapidity of the growth of the tumor, upon its size, and upon how extensive a localized meningitis is present. The diagnosis of the situation of an intracranial tumor from the localizing symptoms is mainly a matter for the expert neurologist. A general view, however, will be given here of the principal localizing symptoms seen with tumors in various parts of the brain.

FIG. 279



Sarcoma of the brain in a boy of 2 years

Cerebellum.—This situation will be considered first because it is the most frequent in early life. Tumors of the cerebellum are more often seen in later childhood than in infancy, and their situation can rarely be definitely localized from the symptoms when they occur in the first two years of life. The chief symptoms of cerebellar tumor are *vertigo* and *cerebellar ataxia.* Vertigo may often be deduced from the movements of the child, even when the patient is unable to describe his sensations. It may sometimes be strikingly demonstrated by placing the child upon its feet and causing it to close its eyes, when a marked swaying of the body will immediately take place and the child may fall to the ground. The ataxia seen in cerebellar tumor is different from that sometimes seen with a lesion of the spinal cord, and often markedly resembles that of alcoholic intoxication. The gait is staggering, and attempts to perform voluntary movements are accompanied by incoördination, which is of a slower type than that seen in chorea and similar conditions. The ataxia may result in complete loss of the power of walking, although there is no muscular weakness. It is notable that paralysis, spasm, and other symptoms of irritation of the motor cortex are usually entirely absent with cerebellar tumor, and that convulsions are rather less frequent than with other forms. The general symptoms of headache, vomiting, and optic neuritis are usually present. The mental symptoms are not present unless there is a secondary hydrocephalus. It should also be remembered that when only one hemisphere of the cerebellum is involved, there may be no local symptoms.

The Pons.—Tumors in the pons are comparatively easy to localize. The diagnostic sign is *crossed paralysis.* This means that the signs of cranial nerve involvement are on one side, and that the motor and sensory symptoms involving other parts of the body are on the other side. When the upper part of the pons is the seat of the tumor, the paralysis is most apt to affect the third and fifth cranial nerves, causing such symptoms as ptosis of the eyelid, dilatation of the pupils, external strabismus, neuralgic pain in the face, and sometimes ulceration of the cornea. There is usually a variable amount of cerebral paralysis affecting the arm and leg on the opposite side of the body. When the tumor is situated in the lower part of the pons, the sixth, seventh and eighth nerves are affected. In such cases there may be a facial paralysis on one side with more or less paralysis of the arm and leg on the opposite side. In addition to facial paralysis, other signs of cranial nerve involvement are internal strabismus, contracted pupils, and sometimes deafness. Convulsions are uncommon with tumors of the pons.

Basal Ganglia.—Tumors in this situation usually do not give any definitely localizing symptoms. When such symptoms are present they depend upon how extensive is the involvement of the various nerve fibers which pass through the internal capsule. A tumor of the basal ganglia may be suspected when there is a combination of motor symptoms in the extremities with symptoms of disturbance in the various tracts of special sense. When, for example, there is a hemiplegia combined with disturbance of the articulation, or of olfactory, auditory or visual senses, a tumor affecting the internal capsule may be suspected. A hemianesthesia is also suggestive of a tumor in this situation. The cranial nerves are usually implicated in new growths situated in the region of the basal ganglia, but in many of these cases the physician is unable to say more than that the tumor is situated somewhere at the base of the brain.

The Motor Cortex.—Tumors in this situation present the easiest problems in cerebral localization, as they have the most definite and uniform localizing symptoms. The principal symptoms are localized spasm or rigidity, localized convulsions and localized paralysis. It is with tumors in this situation that the first manifestation is most often attacks resembling petit mal. These seizures finally change to attacks of convulsions which begin in the face, arm, or leg, but which often rapidly extend so as to become general. These attacks are known as Jacksonian epilepsy. In the attack, consciousness is often retained, or is not lost until late in the attack, after the convulsive movements have become general. The seizures are often followed by a transient paralysis in the face, arm, or leg. The irritative symptoms of a tumor in the motor cortex are rigidity or clonic spasm, involving the face, arm, or leg, according to the situation of the tumor. Usually the most marked involvement is in one of the three situations, face, arm, or leg, but the spasm often involves one of the other situations to a lesser extent. Localized spasm usually precedes the development of paralysis, which is a comparatively late symptom. The paralysis usually first affects one extremity—the arm or the leg—and in some cases the face. Later it may become hemiplegic, involving the entire side of the body.

The Frontal Lobe.—In this situation tumors produce few if any localizing symptoms; in the majority of cases there are none. At times the pressure may extend and cause irritation of the motor area with local or general convulsions, but usually in such cases there is no paralysis. Tumors on the left side of the frontal lobe have sometimes caused apraxia or motor aphasia.

The Parietal Lobe.—In this situation also there are usually no local symptoms. Occasionally with a deeply situated tumor, pressure on part of the optic tract has caused hemianopsia. With involvement of the left inferior parietal lobule, there may, in right-handed persons, be word-blindness.

The Occipital Lobe.—The only constant localizing symptom of a tumor in this situation is hemianopsia. This is usually bilateral, affecting the same side of both eyes, a tumor on the right side of the brain causing blindness in the left half of both eyes, and vice versa. In these cases the patient sees nothing on one side of a line directly in front of him. Sometimes instead of hemianopsia there may be only vague disturbances of vision.

The Temporosphenoidal Lobe.—Tumors in this situation usually have no localizing symptoms. The only diagnostic signs of a tumor in this situation is word-deafness, i.e., inability to understand spoken language.

The Island of Reil.—Tumors in this situation may be latent, or may produce symptoms by pressure upon the motor tract. The only diagnostic sign is seen in some cases when the tumor is upon the left side (right side in left-handed persons). This symptom is motor aphasia.

The Crura Cerebri.—A tumor in this situation, while rare, is characterized by fairly definite localizing symptoms. These are nystagmus, strabismus, and loss of the pupillary reflexes. As the third nerve is most frequently involved, the strabismus is external, and is often associated with dilatation of the pupil and ptosis of the eyelid. With large tumors there is a hemiplegia on the opposite side, and this particular variety of crossed paralysis is quite diagnostic. In addition to the strabismus and loss of pupillary reflexes there may be a varying amount of muscular incoördination and a staggering gait.

The Medulla.—Tumors in this situation are recognized chiefly by the involvement of the lower cranial nerves, glossopharyngeal, pneumogastric, spinal accessory, and hypoglossal. The symptoms are thickened "pudding-mouth" speech, difficulty in swallowing, sucking, or in protruding the tongue, irregularities of pulse and respiration, and vasomotor disturbances. Marked hydrocephalus is often present with tumors in this situation. There may be opisthotonos. Polyuria or glycosuria is sometimes seen. Large tumors may produce paralysis or partial anesthesia from pressure upon the motor or sensory tracts.

The Pituitary Gland.—Tumors of the pituitary gland may be either benign or malignant. Benign tumors do not, as a rule, give any of the characteristic symptoms of intracranial tumor, vomiting and convulsions being usually absent, and headache not a striking symptom. They do, however, give rise to characteristic symptoms which are sometimes referred to as *hypopituitarism* or "Fröhlich's syndrome." In this condition there is a marked increase in the subcutaneous fat with a tendency toward mental dulness, and a notable retardation of sexual development at the age of puberty. Malignant tumors of the pituitary body give localizing symptoms mainly referable to the function of vision. There may be bitemporal hemianopsia followed later by amblyopia. Acromegaly is very rare in early life.

DIAGNOSIS.—Upon a basis of the clinical manifestations alone, no differentiation can be made between cerebral new growths and solitary tubercle of the brain. The physician therefore in distinguishing cerebral tumor from other conditions which it may resemble, must recognize the fact that the tumor may be either a true new growth or a solitary tubercle. After he has arrived at this conclusion, he then turns to the other considerations which enable him to differentiate between these two etiological conditions.

In the consideration of the diagnosis of cerebral tumor, acute conditions, such as acute meningitis, encephalitis and cerebral hemorrhage can at once be excluded on account of the chronic course which characterizes a new growth. The important points in the diagnosis are signs of chronically increased cerebral pressure, particularly the association of attacks of cerebral vomiting, convulsions, headache, and choked disc. The principal conditions entering into the differential diagnosis of cerebral tumor are abscess of the brain, hydrocephalus, tuberculous meningitis, and the chronic stage of epidemic cerebrospinal meningitis.

Cerebral Abscess.—This condition is much less frequent in childhood than is intracranial tumor, and is particularly rare in infancy. In abscess there is usually a history either of trauma or of an infection of the ear, while in tumor there is no definite etiology. Abscess is usually accompanied by fever and leukocytosis, except sometimes in a latent stage. The progress of an abscess is more irregular, with less constancy and severity of the symptoms of headache, vomiting and optic neuritis. Mental symptoms are less marked, and localizing symptoms are indefinite. Involvement of the cranial nerves, and optic neuritis are infrequent. The principal differentiating points, however, are the presence in abscess of septic fever and of a definite etiology.

Hydrocephalus.—This condition frequently occurs as a secondary lesion of cerebral tumor. The recognition of hydrocephalus is easy from the progressive enlargement of the cranium, which is seen in this disease. The question which arises is whether there is a tumor as an underlying cause of the hydrocephalus, or whether the hydrocephalus is due to other causes. When the hydrocephalus is congenital, or when it is associated with some recognizable cause tumor need not be suspected. In other cases the recognition of tumor as an additional factor is extremely difficult.

Tuberculous Meningitis.—Most cases of this disease are more acute in their onset and rapid in their course than cerebral tumor, and consequently do not present much difficulty in differential diagnosis. Occasionally, however, the onset of tuberculous meningitis is very gradual, with a change of the child's disposition, cerebral vomiting, headache, and general or localized convulsions. In these cases the frequent absence of fever increases the difficulty of diagnosis. It should be remembered that tuberculous meningitis is much commoner than is cerebral tumor. The diagnosis depends mainly upon the results of the lumbar puncture, as the cerebrospinal fluid is normal in brain tumor, but characteristic in tuberculous meningitis.

Chronic basilar meningitis in most cases represents a chronic stage of an epidemic cerebrospinal meningitis. It is recognized by the fact that usually there is a history of a preceding meningeal attack. Hydrocephalus, opisthotonos, and rigidity of the limbs are all usually much more marked than in cerebral tumor. An examination of the cerebrospinal fluid obtained by lumbar puncture may easily settle the diagnosis.

Solitary Tubercle.—The physician having excluded other causes which may produce the clinical picture of cerebral tumor must now decide whether the lesion is a true new growth or whether it is a solitary tubercle. The most important thing to remember is that solitary tubercle in early life is infinitely more common as a cause of the symptoms of cerebral tumor than is a new growth. In the last twelve cases at the Infant's Hospital in which the clinical diagnosis of cerebral tumor was made, solitary tubercle was found at autopsy. The recognition of the nature of the process in solitary tubercle depends upon the fact that this lesion is always secondary to tuberculosis in some other part of the body. The recognition of chronic tuberculous infection in early life is often difficult. The factors which enter into the diagnosis have been thoroughly described in the section on Tuberculosis. In general, if d'Espine's sign and interscapular dulness are present, if a roentgenogram of the chest shows enlargement of the bronchial lymphnodes, and if the von Pirquet reaction is positive, a diagnosis of tuberculosis can be made with a fair degree of certainty. In cases of solitary tubercle, if an additional tuberculous meningitis can be excluded by examination of the cerebrospinal fluid, the von Pirquet reaction is usually positive. When a diagnosis of infection with tuberculosis can be made by any of the methods used in the diagnosis of that disease, it is safe to assume that the symptoms of cerebral tumor are caused by solitary tubercle. When all the evidences of tuberculous infection are absent, it is best to assume that the lesion in the brain is a true new growth. When the evidences of tuberculous infection are doubtful, no positive diagnosis as to the nature of the disease can be made, but the chances are in favor of solitary tubercle.

COURSE AND PROGNOSIS.—The prognosis of new growth of the brain is absolutely bad. The progress is steadily downward to a fatal termination. The rapidity of the course is very variable, depending largely upon the character of the growth. Sarcomata are the most rapid form, and with them death may occur in the course of a few weeks. With gliomata the disease may last for many months, and sometimes even as long as two years. Cases have been reported showing all the symptoms of cerebral tumor, in which recovery occurred. It is probable that in none of these cases was the tumor a true new growth. There is a slight possibility of recovery with solitary tubercle, but it is probable that the majority of cases of reported recovery were cases of encephalitis, not tumor.

TREATMENT.—Very little result is to be expected from treatment. The only possibility of cure is from operative interference, and this does not hold out much prospect of success in children. The variety of tumor which is most favorable for operative removal is the endothelioma, which is seen almost exclusively in adults. The tumors in childhood are usually of the infiltrating variety, either gliomata or sarcomata. Sarcoma is rather more favorable for removal than glioma, but the possibility of a total removal of the growth in childhood is very slight. Nevertheless, the prognosis is so absolutely bad that when the diagnosis of cerebral new growth can be clearly made, and when there are symptoms which permit the physician to localize the tumor, operative interference should be considered. The most favorable situation for operative removal is in the cerebral cortex. In any case, a cerebral decompression operation is indicated when the symptoms are severe. This may preserve the sight or other functions of the nervous system for a long time, and also is very effective in many cases in diminishing the pain and general discomfort. Many surgeons do a decompression operation first, and then undertake the removal of the tumor at a later period. I have had a case in which at the time of the decompression operation the tumor was not found, but in which later the tumor grew toward the site of the operation, so that it could be found and removed at a subsequent period. The outlook in children after the removal of the tumor is bad, there being very few recorded cases of complete recovery. Nevertheless, the prognosis of the disease is so absolutely unfavorable that operative interference is justifiable if there is even the faintest prospect of success. In cases with a clear diagnosis of cerebral new growth, I am accustomed to recommend a decompression operation as a palliative measure when the headache is severe, or when other symptoms are marked. I recommend removal of the tumor if it be in any way possible. Except for surgical measures the treatment of intracranial new growth is entirely symptomatic. If there is any possibility of syphilis being present, it is well to give a thorough treatment with iodide of potassium, in order that the remote possibility of the tumor being a gumma may be taken advantage of.

TUMORS OF THE SPINAL CORD

New growths of the spinal cord are very rare in early life. They are almost unknown in infancy, but are occasionally seen in older children, although much less frequently than in adults. It should be remembered that occasionally solitary tubercles and gummata may be situated in the spinal cord and that they give exactly the same symptoms as true new growths.

PATHOLOGICAL ANATOMY.—The new growths may originate from the bone, from the meninges, or from the tissues of the cord itself. New growths originating in the bone are usually sarcomata. Meningeal new growths may be sarcomata, fibromata, or lipomata. The new growths which originate in the tissues of the cord are gliomata or sarcomata.

SYMPTOMS.—The clinical picture of a tumor of the spinal cord is that of a gradually developing transverse myelitis. The first symptom is usually pain, which is referred to the part of the body to which are distributed the sensory nerves arising from the part of the cord involved. According to the situation of the tumor, the pain may be referred to the arms, to the legs, or to the body. When referred to the body, the pain frequently is distributed in a girdle form. The pain may at first be associated with a zone or area of hyperesthesia, and later there may be anesthesia. Motor symptoms usually develop somewhat later, the most common manifestation being spastic paraplegia with exaggerated reflexes, ankle clonus, and later, contractures or tonic spasm. With a tumor low down in the cord there may be paralysis of the bladder and rectum. The tumor is very rarely high enough to cause paralysis of the arms. The most characteristic symptom complex seen in tumor of the spinal cord is the Brown-Séquard paralysis, which is a unilateral paralysis of the leg with a zone of hyperesthesia on the paralyzed side, and with anesthesia upon the opposite side.

DIAGNOSIS.—The diagnosis of new growth of the spinal cord depends upon the gradual development of the symptoms, with the absence of any injury to the spine, or of any caries of the spine. The localization of the new growth is made according to general neurological rules, but it is particularly difficult in childhood on account of the frequent diffuse character of the tumors. Furthermore, the neurological examination of a child is difficult on account of inability of the patient to coöperate with the physician.

PROGNOSIS.—The prognosis is bad. The majority of tumors are of such a character that they either cannot be localized or cannot be removed. Death occurs either from the malignant character of the tumor, or from the infection which results from bed sores and from ascending infection of the urinary tract.

TREATMENT.—The only possible treatment is surgical. If localization and operative removal is impossible, the only available treatment is symptomatic.

IV. FUNCTIONAL DISTURBANCES

In early life the central nervous system is particularly liable to disturbance of function from comparatively slight causes. The etiological factors which act from outside the body in producing disease are the same as those which produce functional disturbance of other organs and systems, particularly various faulty conditions of hygiene and environment. The explanation of the particular tendency of the nervous system in early life to be affected by these etiological factors must be sought within the child. The chief cause is a condition of relatively backward development in those tissues. This condition causes an *instability of the nerve centers* which renders them particularly liable to disturbances of function from causes which otherwise would not be operative. Another constitutional factor, which explains individual variations in the stability of the nerve centers, is *heredity*.

A great many of these functional disturbances cannot be classified under particular disease types. They represent simply symptoms of nervous disturbance. There is no doubt that there is a certain amount of correspondence between the immediately exciting causes and the symptoms. It is only, however, when correspondence between the cause and the clinical picture is particularly characteristic, that we can consider a functional disturbance of the nervous system to represent a disease.

CONVULSIONS

A convulsion is an acute transient disturbance which is characterized by involuntary clonic spasm of the voluntary muscles and by loss of consciousness. The muscles involved may be those of the face, trunk, or extremities, or all of them. Convulsions do not represent a separate disease entity, but rather a symptom of disturbance of the central nervous system, which may be produced by a great number and variety of causes. This disturbance, however, is so extremely common in infancy and early childhood that it deserves special consideration. For this reason I have departed from a strictly etiological basis of classification in order to bring out the various causes which may produce this very common symptom.

In adults convulsions are rare, and usually represent a serious condition with special etiological factors. In infancy and early childhood, on the other hand, convulsions are extremely common, the tendency toward convulsive disorders growing progressively less in later childhood until adult life is reached.

ETIOLOGY.—In the first place, convulsions may be divided into two groups according to whether there is or is not an organic lesion of the central nervous system.

CONVULSIONS DUE TO ORGANIC LESIONS.—In this group belong the convulsions which appear as a symptom of a great variety of cerebral diseases. The pathogenesis is direct irritation of the cortex of the brain. The most common cerebral diseases in early life in which convulsions are seen are:—

1. Meningitis
2. Intracranial hemorrhage
3. Acute encephalitis
4. Solitary tubercle of the brain
5. New growths of the brain
6. Hydrocephalus
7. Cerebral abscess
8. Cerebral embolism
9. Cerebral thrombosis
10. Developmental defects of the brain

Even in this group of convulsions of organic origin, the symptom is more likely to appear in infancy and early childhood than when similar lesions are present in adult life. This can only be explained by certain constitutional factors peculiar to infants and young children. The factor usually held responsible is the instability of the relatively undeveloped nerve centers, and it is probable that this is the chief explanation of the greater frequency of convulsions as a symptom of organic disease. The constitutional disease spasmophilia is, however, so common in early infancy that it may also partly explain the frequency of convulsions at this age, even when the immediate cause of the symptom is the irritation from an organic lesion.

CONVULSIONS DUE TO FUNCTIONAL DISTURBANCE.—The principal etiologic factors which have been connected with convulsions without an organic lesion of the central nervous system are first, *reflex irritation*, and second, *toxic disturbance*. As the same reflex and toxic causes exist in adults as well as in children, there must be other etiological factors to explain the fact that convulsions of functional origin are almost exclusively seen in infancy and early childhood. Until comparatively recently, it has been the prevailing opinion that the chief underlying predisposing cause of the frequency of convulsions in early life was *instability of the nerve centers*, depending upon a relative lack of development of the voluntary centers of the cortex. It must be remembered, however, that convulsions are much less frequent in the first three or four months of life than later, although it is at this very early age that one would expect the greatest instability of the nerve centers. Therefore, while it is very possible that instability from relative lack of development may play a part in the etiology of the convulsions of infancy, it is probable that it does not play the chief rôle, and that we must look further for the principal etiological factor which explains the frequency of convulsions in early life.

Convulsions are most common between the ages of six months and two years. This suggests that the predisposing factor is an acquired one. It is now generally recognized that the commonest cause of infantile convulsions is the constitutional disease, *spasmophilia.* It is known that this disease is characterized by a tendency toward convulsions, and that the immediately precipitating cause of an attack may be either reflex or toxic. Spasmophilia is seen most often between the ages of six months and two years. It is highly probable that the spasmophilic constitution may be acquired without the possibility of its being recognized by the usual diagnostic signs. It is consequently highly probable that in many cases in which the only manifest cause for the convulsions is some reflex or toxic disturbance, the underlying cause is spasmophilia. I believe that the acquired constitutional disease, spasmophilia, is so frequent in infancy and early childhood that it can fully explain the tendency toward convulsions of functional origin which are seen at this period of life. Nevertheless, convulsions are sometimes seen in which at no time can there be obtained any evidence of hyperirritability of the peripheral nerves. In these cases the explanation must lie either in the instability of the nerve centers in early life, or in some special peculiarity of the precipitating cause. In the majority of cases, however, particularly when the precipitating cause is reflex irritation, the underlying cause is spasmophilia.

There remain to be considered the reflex and toxic factors, which, under the modern view of the etiology of infantile convulsions, play the part of precipitating or exciting causes in children who already have a constitutional tendency toward motor discharge from the cerebral cortex.

Any unusual irritation of the peripheral nerves may act as a *reflex* exciting cause. One of the most frequent in infancy is due to the irritation of the mucous membrane of the gastro-enteric tract from the abnormal contents in *indigestion.* While the convulsions associated with infectious processes are usually of toxic rather than of reflex pathogenesis, some *infections* cause a relatively slight toxemia and a relatively marked irritation of the peripheral nerves. Examples of such conditions are acute catarrhal affections of the nose and pharynx, and acute otitis media. It is probable that the convulsions sometimes seen in association with these conditions, particularly in manifestly spasmophilic infants, are of reflex rather than of toxic origin. In rare cases such sources of reflex irritation as *dentition, phimosis,* or *intestinal parasites* have appeared to be the precipitating cause of an attack of convulsions. In the great majority of cases, however, the physician should look either for indigestion or for some acute irritation of the upper air passages.

The commonest immediately exciting cause of *toxic* convulsions is the *onset of one of the acute infections.* Almost any acute infection in infancy and early childhood may begin with a convulsion, although the symptom is most frequently seen in pneumonia, scarlet fever and malaria. The infectious disease which is most often associated with convulsions is pertussis, but here the cause is not manifestly toxic, as the convulsions may be due to cerebral congestion, to slight hemorrhage resulting from a paroxysm, or to the reflex irritation of the lesion in the bronchial mucosa. Second in frequency in the toxic group of precipitating causes is *indigestion with fermentation,* the convulsions being caused by the absorption of toxic products from the gastro-enteric tract. It is therefore evident that indigestion may act in two ways as the exciting cause of convulsions, one being through reflex irritation, the other being through toxic absorption. *Asphyxia* is another exciting cause of convulsions in infancy, and in such cases the effect on the central nervous system is similar to that of a toxemia. *Uremia* is another example of convulsions of purely toxic origin. In rare instances the taking of exogenous poisons may be the cause. While convulsions of toxic origin are commonest in children who are manifestly spasmophilic, yet this underlying cause is not so constantly found as in the reflex group, and it is probable that many forms of intoxication can produce a convulsion without the existence of a spasmophilic factor.

SYMPTOMS.—In the majority of cases the attack comes on abruptly without the slightest warning. Occasionally there are prodromal symptoms, such as restlessness or slight twitchings of some of the voluntary muscles. The first thing usually noticed is that the eyes become fixed, or that the eyeballs are rolled upward. Almost immediately convulsive twitchings begin either in the muscles of the eye, of the face, or of one of the extremities. The twitching extends rapidly until all parts of the body are involved. The convulsion usually becomes general. At times, however, when the cause is a localized organic lesion of the brain, the convulsion may be unilateral, or confined to a single extremity. It should also be remembered that a unilateral convulsion is not diagnostic of a local lesion, but is sometimes seen even in a convulsion of functional origin. The convulsive movements are rhythmic and consist in an alternation of flexion and extension. This produces a series of grimaces in the face and a rhythmic jerking in the extremities. Any variety and combination of tonic and clonic spasm may be seen. The face is pale at first, and later may become slightly cyanotic; the forehead is covered with a cold sweat. The pulse is weak; it may be slow, fast, or irregular. The respiration is shallow, and is often spasmodic. In a true convulsion there is always complete loss of consciousness, and there may be involuntary passage of urine and feces.

The duration of the attack is very variable. In convulsions manifestly due to spasmophilia or associated with reflex precipitating causes, the attack rarely lasts more than two or three minutes. In convulsions due to an organic lesion of the brain, the attack often lasts very much longer, sometimes as long as half an hour, or even more. In the toxic convulsions ushering in an acute infection, the attacks are short, usually not more than two or three minutes, but in certain special cases convulsions of toxic origin may be of longer duration. At the end of the attack ~~the~~ convulsive movements become less frequent, and finally cease altogether, usually leaving the patient in a condition of coma. There may be only one attack, or the attacks may recur after a short interval. Immediate recurrence is not common in spasmophilic and reflex convulsions, but is sometimes seen in convulsions of organic origin, or in certain forms of toxemia, particularly that produced by absorption from the gastro-intestinal canal. After an attack the child soon passes from stupor into a more natural sleep, and unless recurrence occurs, awakens bright and well. A transient paralysis due to exhaustion of the nerve centers is sometimes seen after a convulsion of purely functional origin; this point should be remembered because a permanent paralysis following a convulsion points toward an organic lesion.

Death may occur in a convulsive attack, although this is comparatively rare. Such an occurrence is seen most often in very young infants. While the cause of death may be asphyxia or exhaustion, it is more commonly some underlying factor, such as status lymphaticus, or tetany of the heart. In the great majority of cases recovery occurs, but recurrence at some later period is very common in spasmophilic infants.

DIAGNOSIS.—There is no difficulty in recognizing an attack of convulsions. The recognition of the cause, which is important for purposes of intelligent treatment, is often a matter of extreme difficulty. The physician must obtain a careful history, not only as to the character of the immediate attack, but as to all other symptoms which may have been present. A most careful and thorough physical examination must be made.

It is particularly difficult to determine the cause of convulsions occurring in the early weeks of life. At this period convulsions may be due to such organic lesions as cerebral hemorrhage or defective cerebral development without showing any localizing symptoms. It is possible that the mechanical disturbance of prolonged labor may give rise to convulsions through circulatory disturbance without the existence of an organic lesion. While spasmophilia can be excluded at this period, convulsions of toxic origin from gastro-intestinal disorder are a possibility. In the majority of cases, however, at this age the cause is organic.

ORGANIC BRAIN DISEASE.—This cause should be suspected whenever the convulsive attack is prolonged. The diagnosis depends mainly upon whether a careful and thorough physical examination reveals any focal symptoms, such as a localized spasm or paralysis, strabismus, or changes in the pupils. Limitation of the convulsions to one side or to one limb, while strongly suggestive of an organic lesion, is occasionally seen in purely functional disturbance. Convulsions due to an organic lesion of the brain are not accompanied by fever, except in acute meningitis and encephalitis. In hemorrhage and meningitis the convulsions are likely to recur within a short time. In solitary tubercle and new growths they usually recur after a much longer period.

SPASMOPHILIA.—This is the commonest cause of convulsions in infancy and early childhood, and should always be suspected when there is no evidence of an organic lesion. The diagnosis depends upon the recognition of such diagnostic signs as laryngospasm, carpopedal spasm, Chvostek's sign, the peroneal reflex, and Trousseau's sign. The diagnosis is often impossible in the period which immediately follows the convulsive attack, but if a careful examination be made on each of the successive days after an attack, some one of the characteristic signs will usually be found if spasmophilia be present. The recognition of spasmophilia does not preclude the possibility of a reflex or toxic exciting cause.

TOXIC CAUSES.—The most important point in the recognition of the onset of some acute infection as the cause of convulsions is the existence of a high temperature. The only organic brain diseases accompanied by fever are acute meningitis, acute encephalitis, and cerebral abscess; and these conditions can usually be recognized by their characteristic symptoms. Uremia, while a rare cause of convulsions in early life, is a possibility, and an examination of the urine should be made in all cases in which the origin of the convulsions is not clear. Asphyxia is a possibility as a cause in the convulsions occurring in the newborn, and in rare cases produces convulsions late in the course of a pneumonia, or in membranous laryngitis.

REFLEX CAUSES.—I am very doubtful if reflex irritation ever causes convulsions except as a precipitating cause in spasmophilia.

It is always well, however, at the time of a convulsion to consider the possibility of the child being spasmophilic and that the precipitating cause is indigestion, and the immediate treatment of the case often depends upon this assumption.

EPILEPSY.—This possibility should only be considered when all other causes have been excluded. It is the least probable diagnosis in infants. In older children it is recognized mainly through the existence of a distinct aura preceding the attack, or by the existence of a history of previous typical attacks of petit mal.

PROGNOSIS.—This depends entirely upon the cause of the convulsions. In general, convulsions in themselves are rarely fatal unless they occur as a terminal condition in some recognized disease. Fatal attacks are seen most often in the newborn or in very young infants, in whom the cause is obscure. They are occasionally seen in pertussis, or late in the course of laryngeal or pulmonary disease accompanied by asphyxia. When the convulsions are prolonged and repeated, the evidence points toward a grave lesion as the cause, and the outlook is doubtful. In convulsions which can be definitely associated either with spasmophilia or with the onset of an acute infection, the prognosis is good in so far as the convulsions are concerned. It is rare that a convulsive attack is in itself the cause of any injury to the brain, although I have seen a case in which convulsions were produced by atropin poisoning and were followed by the symptoms of cerebral hemorrhage. In the great majority of cases a convulsion means only some nervous hyperirritability. The possibility of epilepsy is to be considered in children over two years old, in whom convulsions without apparent cause recur after varying intervals.

TREATMENT.—The physician who is summoned to attend an attack of convulsions should be prepared with the following things: Ether, a hypodermic syringe with morphin, a solution of chloral, some sodium bromide, and the soft rubber catheter, tube and funnel, which are used for the giving of a high irrigation of the colon or for washing out the stomach. In the majority of cases the infant will have been rushed at once into a warm bath, and I find that my first duty in such cases usually is to see that the water of the bath is not too hot. It is well to order that the infant be taken out of a hot bath before it returns to consciousness in order that a recurrence of convulsions from fright may be avoided. If the infant has not been placed in a warm bath, I usually substitute a mustard pack, given as described in Volume I, page 157. The patient should be kept perfectly quiet, every unnecessary disturbance being avoided. Cold may be applied to the head and dry heat to the feet, or the feet may be placed in mustard water while the child is lying in the mustard pack. When the convulsion is only of a few minutes duration and is not repeated, no medication need be used. In infancy, however, it is well to give a thorough irrigation of the colon in order to remove any possible source of reflex irritation in the lower bowel. If the history suggests the possibility of undigested food in the stomach being the exciting cause, gastric lavage should be performed. The source of irritation, however, is more commonly in the intestine, and after the irrigation of the colon I usually give the patient a dose of castor oil as a prophylactic measure against the possibility of recurrence. When the physical examination reveals a high temperature suggesting that the cause of the convulsion is the onset of an acute infection, recurrence can best be prevented by the use of the cold pack.

In prolonged convulsions additional measures are necessary. The most effective remedy is an inhalation of ether, and this should be given whenever the convulsion continues after the initial measures of counter-irritation are instituted. If the child upon coming out of ether tends to relapse at once into a prolonged convulsion, chloral should be given by rectum in doses proportioned to the age of the child. (See Volume I, page 170). The chloral should be injected as high as possible into the bowel through the soft rubber catheter, and its escape should be prevented by pressure of the buttocks together. If the convulsive movements are very severe, while the physician is waiting for the chloral to take effect, a second ether inhalation may be given for temporary effect. If after twenty or thirty minutes the chloral has not taken effect, or if it has been expelled from the rectum, and if the convulsion tends to continue as soon as the ether is withdrawn, morphin should be given hypodermically in doses proportioned to the age of the child. If in spite of all these measures the convulsion still tends to continue, the giving of chloral by rectum may be repeated once, and if this fails, an injection of morphin can be once repeated after the expiration of two hours. While the physician is thus attempting to get a prolonged convulsion under control with chloral or morphin, he should use the ether inhalation often enough to prevent undue exhaustion of the child, even though its effect is only temporary. When such a prolonged convulsion is finally under control, recurrence should be prevented by giving sodium bromide by rectum in doses proportioned to the age of the child. (Volume I, page 170.) If, in spite of the bromide a severe convulsion recurs, the treatment of the original attack should be repeated.

In some cases, the condition against which the physician has to contend is not a prolonged, severe convulsion, but a tendency toward the frequent recurrence of shorter convulsions at various intervals. This condition is best met by the use of sodium bromide given by rectum. If this is ineffective in preventing the occurrence of repeated convulsions, chloral in proper doses should be added to the rectal injections.

As soon as the convulsions have been brought under control, every effort should be made to find and treat the probable cause. Indigestion should be combatted by appropriate reduction in the strength of the food given. In cases of repeated convulsions apparently due to the absorption of the products of gastro-intestinal fermentation, I have found lactic acid milk the most useful dietetic resource. Some cases of obstinate convulsions yield to inhalations of oxygen better than to any other measure, particularly when the cause is asphyxia, or when the convulsions occur in early infancy.

EPILEPSY

Epilepsy cannot be considered a very sharply defined disease either upon the basis of etiology, of pathological anatomy, or of symptoms. It is usually considered a symptom complex in which a great variety of causes may be operable. As the principal symptom of epilepsy is a general convulsion which closely resembles the convulsions of spasmophilic or toxic origin which are so common in infancy and early childhood, it might seem at first that there is no essential difference between epilepsy and these infantile convulsions of functional origin. Nevertheless, epilepsy shows certain peculiarities which suggest that it represents a different process from ordinary infantile eclampsia. True epilepsy differs from the convulsions which were described under the preceding heading in the following particulars: In the first place, while attacks may begin in infancy, they are much more likely to begin at a later period of life, particularly about the age of puberty. A still more important difference is that the tendency toward convulsions produced by reflex or toxic exciting causes is confined to infancy and early childhood, and there is no tendency toward a repetition of the convulsive attacks in later childhood and in adult life. Epilepsy, on the other hand, is a disease in which the seizures not only tend to continue throughout life, but also to grow worse with the lapse of time. A third very important difference lies in the fact that the seizures in epilepsy do not ever appear to be associated with any particular exciting cause, but occur, as it were, out of a clear sky, without any connection with either reflex or toxic disturbances. There is still another difference in the nature of the attacks. While both conditions are characterized by general clonic convulsions with loss of consciousness, in epilepsy there is often a peculiar aura which precedes the attack, and there are also seen certain peculiar mental states which have been described under the term "equivalents." It is these differences which chiefly entitle epilepsy to consideration as a diseased condition which differs in its essential nature from the other forms of convulsive seizure seen in early life.

It is more difficult to draw a distinction between epilepsy and the convulsions which are symptomatic of definite organic disease of the brain. We have seen that such organic lesions of the brain as encephalitis, an old hemorrhage, and cerebral tumor, may be accompanied by epileptiform convulsions with loss of consciousness, and that these attacks may be repeated at various intervals. Furthermore, in these conditions the convulsive seizures may not cease with the expiration of early childhood, but may continue to occur at intervals in later childhood and in adult life. Attacks due to these recognized lesions, moreover, resemble those of epilepsy in that they are not associated with reflex or toxic precipitating causes. The attacks may even be preceded by an aura, and in patients with cerebral tumor, mental states are sometimes seen which are precisely like those observed in epilepsy. In fact, from the symptomatic point of view, no distinction can be drawn between true epilepsy and the so-called symptomatic epilepsy which may occur in a great variety of organic diseases of the brain. The only difference is one of etiology and pathological anatomy. Most cases of "symptomatic epilepsy" are due to a definite recognizable cause, and are associated with a constant easily demonstrable anatomical lesion of the brain, whereas in true epilepsy the cause is entirely unknown and demonstrable anatomical lesions are absent. True epilepsy therefore may be defined as a disease characterized by a tendency to repeated attacks of general convulsions, which differ from the common infantile convulsions of functional origin in their tendency to recur throughout life, in the absence of precipitating and exciting causes, and in the presence of an aura or of peculiar mental states, and which differ from the convulsions of organic origin in the absence of a definite etiology and of constant anatomical lesions of the brain.

ETIOLOGY.—It is usually granted that the initial disturbance of true epilepsy originates somewhere in the cortical centers of the brain, and that the epileptiform convulsion results from the liberation of energy from the cells in the motor cortex. The most modern view regards epilepsy as not representing a purely functional disturbance, but as representing the effects of a lesion of the cerebral tissues, which acts as a source of irritation and produces the occasional explosion or motor discharge which characterizes the disease. The evidence in favor of this hypothesis lies in the fact that in many cases of true epilepsy microscopic lesions have been discovered, although the nature of these lesions has not been constant. The most important associated etiological factor which has been established is *heredity*, this appearing to play the most important rôle in the production of the disease. It has been estimated that at least 50 per cent. of epileptics come from epileptic families. This fact would suggest that the lesion which produces the irritation is more likely to be of developmental origin than acquired from some influence acting from outside the body. It is quite probable that many if not the majority of cases of epilepsy are due to such a developmental lesion, which often cannot be demonstrated by our present methods of examination. Nevertheless, there is evidence that acquired lesions, such as encephalitis or hemorrhage, may occur, which are too small to produce the paralytic and other localizing symptoms associated with larger acquired lesions of this kind, but which nevertheless may leave behind a tiny area resembling a scar which might prove a constant source of cortical irritation, and which would produce the same effects as a developmental lesion. It is possible that the cases of epilepsy in which heredity does not appear to be a factor are due to acquired lesions of this kind. Evidence that epilepsy is due to the presence of an organic lesion, whether developmental or acquired, is constantly accumulating.

The influence of other conditions, such as alcoholism in the parents, and syphilis, has not been definitely established in connection with epilepsy, although there is some evidence that these factors may lead to a microscopic lesion of the brain, which would account for the existence of the disease. An innumerable number of possible toxic and reflex causes have been suggested, but the weight of evidence inclines toward the view that there is no proof of the influence of these factors.

The question has frequently been raised whether infantile convulsions are not frequently followed by epilepsy in later years. The answer to this question appears to be negative. It is true that epilepsy may begin in infancy, but in the majority of cases of this kind there is no evidence of spasmophilia, which is the commonest cause of infantile convulsions, nor is there evidence of a gross recognizable anatomical lesion of the brain, or of definite reflex or toxic exciting causes.

PATHOLOGICAL ANATOMY.—No constant or characteristic lesion has been found in cases of so-called idiopathic epilepsy. Nevertheless, it is the opinion of the most competent authorities that a pathological tissue change can be demonstrated in the majority of instances. These changes can only be observed microscopically, and the character of the changes described by different authorities differs widely. Some investigators have described a generalized gliosis, others, areas of sclerosis in various parts of the cortex, while still others have observed degenerative changes in the ganglion cells. It is probable that epilepsy is caused by some small focal lesion which is either developmental in origin, or acquired, and that the symptoms are due rather to the presence of such a lesion than to anything peculiar in its character. It is thus conceivable that a great variety of microscopic lesions might produce the disease.

SYMPTOMS.—Epilepsy may begin in infancy or at any time throughout childhood, but a frequent time for its development is at puberty.

PETIT MAL.—The *petit mal* may exist in different degrees of severity. In the mildest form, which may often pass unnoticed unless the attendants are especially on the watch for it, the child stops for a moment in its occupation, whether speaking, eating, or playing, while its eyes become fixed, the face blanches or becomes congested and assumes a vacant expression. This condition may last for only a few seconds, when the child resumes its former occupation as though it had never been interrupted, and usually is not aware that anything has happened. In other cases this condition lasts a little longer, and slight twitching of the lower part of the face and of the extremities may occur. Again, the attacks are more severe, the child complains of being dizzy, staggers, has slight convulsive movements, and turns pale, this condition lasting for a minute or more, and being quite marked, but without any total loss of consciousness. Momentary attacks of staggering sometimes occur alone in place of the attacks above described. At times these attacks of petit mal are the only manifestations of the disease, but in severe cases they are apt to be accompanied by occasional attacks of grand mal. They may occur as often as twenty or thirty times a day, or, on the other hand, they may be noticed only once in four or five days, and sometimes they are absent for longer intervals.

GRAND MAL.—In the *grand mal* the attacks are of much greater severity. They are sometimes preceded for several hours by a feeling of malaise or general discomfort, but this is not always present. Patients sometimes have notice of the sudden onset of the attack, experiencing a sensation which immediately precedes the convulsion and forms part of the attack itself. This is called the *aura*. This aura may be of different kinds. It is most commonly a sense of fulness or oppression in the epigastrium, from which something seems to rise into the throat, and unconsciousness supervenes. It may be, however, a pain or a sensation of numbness, tingling, or other form of paresthesia in various parts of the body. Sometimes tinnitus is the first symptom. Frequently the patient has no warning whatever of the attack, but falls unconscious with or without a cry. The face becomes congested, and the eyes usually turn upward so that only the whites can be seen. After this follows the stage of tonic spasm, which is sometimes so short that it is overlooked. Then come the clonic convulsions, which in typical cases are general, although the limbs on one side of the body are sometimes more affected than those on the other side. In such cases a post-epileptic hemiplegia lasting several hours or days may result. The movements of the limbs are apt to be very violent, the hands are clinched, the thumbs being fixed on the palms and the fingers closed over them. In many cases the patients froth at the mouth. In the more severe cases the children bite their tongues and pass their urine involuntarily. The duration of such attacks is usually five or ten minutes, but one attack may succeed another with little or no intermission. When the attacks follow one another in this way for several hours the patient is said to be in the *epileptic status*, and this condition as regards life is very serious. After the convulsion ceases the child's breathing becomes stertorous and the limbs are relaxed. Later, and before consciousness fully returns, the child often falls into a deep sleep, and on waking has no recollection of the attack, but complains only of headache and of mental confusion. Attacks often occur in the night, and in this way may be overlooked, the only evidence of them being that the child has wet the bed. In certain cases, when only nocturnal attacks have been present, we often have reason to believe that the disease has existed for a considerable period before its presence was suspected. In some cases in connection with the attacks there is a desire to walk or to run, so that the patient must be closely watched. In this condition children may walk straight against an obstacle, though they are more apt to stop when something comes across their path. Sometimes they walk or run in circles.

The cases of paroxysmal running described by Bullard are at times early manifestations of an epilepsy which will develop later, although they may also be only the symptoms of hysteria, chorea or organic cerebral disease.

In the so-called *massed form* of epilepsy, the convulsions occur at frequent and at very short intervals, then cease suddenly only to recur again after an interval of many months in the same massed form. In later life the attacks eventually become more evenly distributed and assume the usual type of grand mal. This variety is very typical in the beginning and may give rise to a suspicion of hysteria. It is peculiarly resistant to treatment. Epileptic children are liable to bursts of ungovernable anger and violence lasting for hours, in which they may tear and destroy things, bite the mother or nurse, and are apparently for a time under the influence of illusions and hallucinations. This form is called *psychic epilepsy* in distinction to motor epilepsy, and is due to the nervous explosion taking place in the psychic instead of in the motor areas. The motor attack may precede the psychic, or vice versa.

The condition of patients between the attacks is in the lighter cases and in the beginning of the disease usually quite normal. As the disease progresses, however, there is a tendency to mental impairment, and in the more severe cases, in contrast to the lighter ones, we are apt to find some enfeeblement of intellect, which at times may go on to an advanced dementia. Such cases are to be distinguished from the symptomatic epilepsy seen in cases of defective mental development.

DIAGNOSIS.—As the convulsive attacks occurring in epilepsy cannot be distinguished clinically from similar attacks due to other causes, we are forced to differentiate epilepsy from other diseases by carefully eliminating other causes for the convulsions. We must also wait to see whether the attacks will continue indefinitely, in which case they are more likely to be epileptic.

The diagnosis of epilepsy is made from a continuance of the attacks after a considerable period without evidence of any organic disease or toxic or reflex irritation. When the child bites its tongue during the attack and goes to sleep after the convulsion, or when there is temporary mental impairment after the convulsion, we have good reason to state that the convulsions are due to true epilepsy, especially if no symptoms of organic brain disease coexist.

Epileptic are easily distinguished from hysterial convulsions by the presence of consciousness in the latter, at any rate to a considerable extent. Hysterial convulsions in children are not very common, and almost never exist without the presence of other symptoms of hysteria.

PROGNOSIS.—The prognosis of epilepsy as to life is, on the whole, favorable, and epileptics may live for years.

As regards cure, the prognosis in cases beginning in early infancy is very serious but by no means hopeless. When the disease begins at the age of ten years or later a certain number seem to recover, at least temporarily. Many authorities consider that true epilepsy is never cured, yet undoubted cases exist in which no convulsions take place for years.

The prognosis is influenced by the length of time the disease has run before thorough treatment is instituted. If begun early and carried out systematically for months or years according to the severity of the case, the chances of complete recovery are greatly increased. Death may occur from accidents during an attack, such as falls, being smothered in bed or drowned while bathing.

TREATMENT.—The child should be treated at once, in order to avoid continuous shocks to its nerve centers. Much benefit results from early attention to general hygienic conditions and to protection from nervous disturbances. The management of these cases demands constant watchfulness and tact, so as to regulate the surroundings of the child in such a way as to avoid all sources of nervous excitement and irritation, such as arise from improper diet, gastric and intestinal disorders, genital irritation, and adenoid growths. The diet must be regulated according to the especial indications for each patient. A vegetable diet is usually indicated, but when the child does not thrive on this it is advisable to give a certain amount of meat. Eggs are usually well borne.

The bromides in some form are the most useful drugs. At present there is a reaction against the absolute exclusion of nitrogenous food. It should, however, be given in limited quantities. Of far more importance is the diminution or exclusion of chlorides, especially table salt, from the food. It has been demonstrated experimentally and clinically that bromides exert a much more beneficial action when chlorides are excluded and considerably smaller doses are required to produce the same effect. The untoward results of the bromides are also less likely to develop. In severe cases sodium bromide may replace the salt in cooked food and be used at the table. One drachm to one loaf of bread is the amount usually used. When such substitution is made it is important to keep an accurate record of the amount of bromide administered in this form. Tourette has suggested a very efficient combination in the proportion of two parts of potassium bromide to one part each of sodium bromide, ammonium bromide and sodium benzoate. It is best to begin with a dose of 6 grains three times a day for infants of one to two years, and to double this amount for older children. If the convulsions are not controlled, the doses should be increased gradually until the physiological action of the drug is noticed. This treatment should be carried out for long periods, but under constant supervision, and from six months to a year after the convulsive attacks seem to have ceased.

HYSTERIA

Hysteria is a functional disturbance of the cerebral centers. According to the theory of Janet, it depends upon a dissociation of consciousness and subconsciousness. Impulses originate subconsciously, which, lacking the inhibitory influences of the higher function of consciousness, lead to most diverse mental and physical conditions. The name is a misnomer, in that the disease has no necessary connection with uterine disorders, but it has been generally adopted.

ETIOLOGY.—We know very little about the etiology of hysteria. Well-marked instances of the disease occur in early life, usually in the middle and later periods of childhood. Oppenheim states that he has repeatedly observed hysteric symptoms in children from two to three years of age. It is, however, a rare disease in early childhood, although many cases begin at puberty. An inherited nervous organization or highly exciting surroundings, combined with a lack of proper home discipline, appear to be the most important factors in the development of the condition in children. The direct exciting cause may be anything producing mental or physical shock in one who has by inheritance a neuropathic predisposition. Events or associations calling up from the subconsciousness memories which have been forgotten by the consciousness may precipitate an attack of hysteria. The disease is much commoner in girls than in boys.

SYMPTOMS.—The mere presence of emotional or imaginative conditions in children does not constitute hysteria. For the existence of the disease it is necessary to have definite symptoms, either a markedly disorganized mental state, paralysis, anesthesia, or some serious loss of function.

The symptoms are innumerable. Convulsions and paralysis are common, while dysphagia, amaurosis, deafness and anesthesia are met with only in very severe cases. Anesthesia is especially interesting as representing a pure type of the disease, and is usually on one side of the body, hemianesthesia, or it may be of the "glove" or "stocking" type, the anesthesia not corresponding to any distribution of peripheral nerve or spinal cord lesions. The anesthesia may extend to the organs of special sense leading to partial or complete loss of the function of hearing, sight, smell or taste, but the last two are hard to determine with accuracy in early life.

Hysteria in children as usually seen in America is marked by the emotional condition of the child, and by the presence, in many cases, of a fixed idea relating to its own physical condition. The child subconsciously performs certain actions or functions which consciously he cannot control. There probably has often been in the beginning some real difficulty or disturbance of the performance of these functions, such as pain, which has passed away or which is not sufficient to produce the present condition.

The most common symptoms, aside from the mental condition, are *convulsions*, *paralysis*, *anesthesia* or *hyperesthesia*.

The *convulsions* are distinguished from those of epilepsy by an alteration rather than a loss of consciousness and by the exaggerated character of the movements. The patients may or may not be consciously aware of their actions and surroundings, but subconsciously, as shown by hypnosis, they always know what is taking place. In epilepsy the loss of consciousness is complete and the memory of the event cannot be aroused in the subconsciousness. If a patient is not actually seen in an attack the diagnosis from epilepsy may be difficult, but the presence of one or more of the stigmata of hysteria, that is of anesthesia, hyperesthesia, muscular spasm or abulia, gives presumptive evidence of the hysterical nature of the convulsion. The patient rarely seriously injures himself in falling, and does not bite his tongue. He does not sleep after the attack but is often dull and apathetic.

The *paralysis* is often of the spastic form, and may be either hemiplegic, paraplegic, or monoplegic. In this form the limbs are rigid and the knee-jerks are exaggerated. It may, however, be of the flaccid variety, with the knee-jerks diminished or absent. It is distinguished from the organic forms of paralysis by the normal reaction of the muscles to electricity, by the absence of atrophy, by the absence of any affection of the sphincters and at times by the presence of anesthesia. When *anesthesia* occurs it is usually irregular in distribution, occurring in patches, but at times it suggests the distribution seen in cerebral organic disease. In organic disease the anesthesia generally grades off, whereas it is sharply outlined in hysteria. It is often variable, changing more or less from day to day. *Aphonia*, which was originally dependent upon an acute laryngitis, may persist for months as an hysterical condition when the organic cause has ceased to exist. In a similar way, inability to walk because of an acute rheumatic fever affecting the knee-joint may be followed by hysterical paralysis of long duration. If a patient is seen for the first time in the state of hysterical paralysis, one may, by putting her into a condition of hypnosis and questioning the subconsciousness, learn the association of ideas which has given rise to the paralysis. By suggestion in the hypnotic state and explanations in the conscious state the idea may be dispelled and the hysterical attack relieved. The anesthesia so characteristic of hysteria illustrates the dissociation of consciousness and subconsciousness. There is no conscious sensation to touch or pain during its existence, but if the patient is hypnotized and questioned it may be clearly demonstrated that touch has been perceived subconsciously.

We sometimes meet with exaggerated hysteria in children. Such attacks are characterized by screaming, running, jumping, and a feeling of being pulled about. They may last for hours, or for days. The duration of the type, however, is usually long—at times, with intervals, over a year. No signs of organic disease are found in these cases; the patients seldom injure themselves, and are finally cured by moral influence, change of scene, and good hygienic surroundings.

Hysteria occasionally causes children to present symptoms of serious disease of the spine or joints. This most often follows some slight injury, but may occur spontaneously.

DIAGNOSIS.—The diagnosis of hysteria is made first by the exclusion of organic disease, and then by the presence of a combination of symptoms which include many diverse conditions which are not usually associated in organic disease. The history of a neuropathic predisposition is of value when obtained in a case which shows the symptoms described. The absence of the reaction of degeneration in hysteria enables us to differentiate flaccid hysterical paralysis from that of anterior poliomyelitis. Generally the diagnosis is not so difficult as in adult life, as the child's mind is less experienced, is less subject to fixed ideas, and is, therefore, not so antagonistic to suggestion.

PROGNOSIS.—The prognosis in cases of hysteria in children is, as a rule, favorable, but recurrences may take place in later life.

TREATMENT.—Hysterical children are peculiarly susceptible to treatment by suggestion, but their management must be placed in the hands of some one with tact and firmness, who appreciates the difference between hysterical symptoms which are not under the control of the child's consciousness, and similar conditions which are often due to bad temper or malingering. Regulation of the child's life and environment upon the principles of rational diet and hygiene, especially the avoiding of excitement and over-exertion of all sorts, whether from work or play, are of the first importance in prophylaxis. The confidence of the child must be gained by encouragement and education. It must be taught and made to overcome and control the disturbed functions. This is best accomplished by those who are especially experienced in mental therapeutics. Hypnotism is justified only when other methods fail. Static electricity and hydrotherapeutics are sometimes useful adjuncts to moral treatment.

CHOREA

(Sydenham's Chorea; St. Vitus's Dance)

Chorea is a disease characterized by irregular and involuntary muscular movements without loss of consciousness, and affecting the muscles of volition.

The disease is rare in infancy, but may occur in the early months of life. It seldom begins after puberty. It is most apt to begin and is most marked in its symptoms during the period of the second dentition — that is, during the period of active growth, from six years to puberty. The greatest number of cases is found among the female sex and among those who do not receive sufficiently nutritious food. A sharp distinction should be made between the disease chorea, with its characteristic choreiform symptoms, and the same choreiform symptoms resulting from other diseases, sometimes represented by central nervous lesions, sometimes by purely reflex causes. It will save much useless reading of the literature of chorea and much profitless discussion as to its etiology and pathology if this distinction be borne in mind. Eliminating those cases showing choreiform movements which are due to gross lesions of the nervous system, such as the post-hemiplegic and congenital forms, we can at once very materially reduce the number of cases of true chorea. In like manner we should separate from true chorea those cases of peripheral irritation in which the partial choreiform symptoms are evidently reflex and can be cured by removal of the cause. Examples of these reflex choreiform symptoms are the facial chorea from nasopharyngeal irritation and the partial choreiform movements occasionally arising from errors of refraction and ocular insufficiency. The consideration of these anomalous forms of chorea belongs with the diseases in which they occur.

ETIOLOGY.—Chorea can occur in connection with other diseases, such as measles, scarlet fever, and typhoid fever, although this, in my experience, rarely occurs, and even then chiefly among the poorly cared-for. A certain number of cases have so directly followed intense fright that we must acknowledge acute mental conditions as a possible cause. The disease which is most frequently associated with chorea is *rheumatic fever*. The percentage of cases, however, in which this association takes place is difficult to determine. This difficulty arises from the want of uniformity in the reported cases of different observers, due to their different ideas as to what constitutes rheumatic fever. The question of the bacteriology of chorea will be discussed under Problems and Research.

The association of chorea with tonsillitis and rheumatic fever is so notable, and cases of chorea so frequently follow closely or alternate with attacks of rheumatic fever, that there is considerable reason for believing that chorea is in certain cases one of the manifestations of this infection. The frequent association of chorea with signs of endocarditis, with the fact that the great majority of cases of endocarditis are due to the rheumatic fever infection, is further evidence of the rheumatic origin of chorea. There is no definite proof of the opinion held by some authorities that chorea is *always* due to rheumatic fever infection. It is possibly a particular nervous manifestation due to a variety of causes, of which rheumatic fever is one. Overwork at school during the first part of the school year has so often been shown to result in an attack of chorea in the spring and in a recurrence in the autumn on returning to school, that it should be recognized in considering the different causes of the disease. This may act, however, by making the child more susceptible to infection, the chorea being due to infection. Strain of the ocular muscles has been considered an exciting cause of chorea, but, beyond producing in some cases choreiform symptoms, it cannot be considered as a cause of true chorea. Any local causes of irritation, such as adenoids or parasites, and such conditions as bad hygiene and improper food, may be contributing causes.

An hereditary neuropathic predisposition has long held a prominent place in the etiology of chorea, but in my opinion is not important.

PATHOLOGICAL ANATOMY.—There is a large number of cases of chorea in which the disease is found to have no apparent pathological lesion. The symptoms, however, show us that the morbid process is located in some part of the central nervous system. Whatever the nature of the lesion, it gives rise to a profound excitement of the motor centers, presumably due to their inanition, and is accompanied by a temporary inability of these centers to recover themselves. Many lesions have been described as occurring in chorea, but in the pure cases (Sydenham's chorea) of which we are speaking, which really represent the disease, there is no lesion which with our present knowledge we can say is characteristic.

SYMPTOMS.—Chorea may be in its distribution general or partial; in its course, acute, subacute, or chronic. In many cases the disease is exceedingly mild in its symptoms and is of a benign type; in others it assumes a severity which seems to threaten life. The beginning, although at times sudden, as from fright, is, as a rule, gradual, a few muscles only being affected at first. The child becomes fretful and impatient. Its irritability must be carefully differentiated from bad temper, for which the symptoms are apt to be mistaken by the family. The clinical picture of the disease is a jerky, irregular, involuntary, and purposeless contraction and relaxation of the muscles, which usually begin in the fingers, hands, shoulders and face. There is an irregular, uncertain action of the part affected, and efforts of the will only partly control the movements. A notable tremor of the tongue when protruded, and of the fingers when the hand is extended, is a very constant and characteristic sign of chorea, which is particularly valuable in the diagnosis of the milder cases. As the disease progresses, the voluntary control of the muscles diminishes more and more, and at times disappears entirely.

The movements ordinarily cease during sleep, but in severe cases they continue and even interfere with it. The skin may be worn off of prominent portions of the body by friction, especially on the knees and elbows. At times the child is unable to walk, on account of incoördination. The speech may become slow and indistinct, from the affection of the muscles of the tongue and of the larynx, and even mastication and deglutition may prove difficult. In very severe cases the difficulty in speech may be enhanced by the mental condition, which may be represented by dulness and apathy, and the child cannot or will not speak. The tendon reflexes are lessened in severe cases. The muscles become weak and soft, and there is considerable emaciation. There is usually loss of appetite, and the bowels are often constipated. The dynamometer usually shows impaired muscular power. In certain cases the muscles of the extremities on one side of the body are principally or alone affected (hemichorea). These cases do not differ from the ordinary bilateral cases in any way except in this respect.

The choreiform movements are so prominently a feature of the disease that one may underestimate the seriousness and extent of the mental symptoms, which often persist after the muscular incoördination has subsided. The nature of the child in very severe cases seems to change; irritability, disobedience, impairment of the powers of memory and application, and spells of crying and general nervous excitability may be the early manifestations of a condition of mental derangement which subsequently give rise to delusions, hallucinations and even mania. Cases have been reported of this nature which have terminated fatally. In some cases there may be involuntary evacuations of the feces and urine. The disease is distinct from epilepsy, and there is little danger of the patient becoming epileptic unless it happens to develop in an individual who is predisposed to that condition.

PROGNOSIS.—Chorea is very apt to show relapses and to recur for several years. Although often obstinate in the persistency of its symptoms, yet it may be said to be self-limited, and, as a rule, to recover, provided no complications, such as of the heart, arise. The time which elapses before complete recovery is very variable; mild cases may recover under proper treatment in six weeks, but the severer forms usually extend over a period of three or four months in spite of all efforts at treatment. Although chorea is ordinarily considered a benign disease, yet we must always look upon it as a serious disturbance until we are sure that we are dealing with the usual mild form of the affection.

We must allow that even uncomplicated chorea is a varying disease as to the severity of its symptoms and their persistence for a longer or a shorter time. We also know that there is a marked tendency to relapse, and that the number of relapses varies to a great degree. The length of the attack and the response to treatment may differ much. Bearing these facts in mind, we can understand the rapidity with which certain individuals are attacked or the quickness with which they recover. Some cases recover rapidly under only hygienic treatment, while others are apparently unaffected by any drug whatever. When heart-murmurs, evidently representing organic disease, appear, it is often a cause for comment at the comparatively slight discomfort which the cardiac lesions entail. At times, again, it is surprising how rapidly fatal are some cases which are complicated by cardiac disease, and how uncontrolled they are by any treatment whatsoever. I have seen well-marked cases of chorea get well in from sixty to seventy days where good food and a small amount of tonic (nux vomica) constituted the entire treatment.

TREATMENT.—Our main reliance must be placed upon regulation of the child's life in such a way as to provide rest, freedom from excitement, fresh air and nutritious food. Disturbances in the function of digestion and excretion must be treated symptomatically. If extreme muscular incoördination exists, absolute rest in bed and even restraint is required. In the average case the child should be kept constantly in bed until all marked symptoms have subsided. Cold packs are too severe for the majority of cases, and warm sedative baths are much to be preferred. Skilled nursing is important in the treatment in carrying out the routine life outlined and in protecting the patient from diversions and annoyances which aggravate the disease.

It is especially important to keep the child away from all forms of diversion and excitement. There must be no contact with other children, and it is best to use complete isolation, the entire care of the child being taken by a trained nurse.

It may be necessary to pad the bed and apply light splints to control the movements of arms and legs during sleep and to guard against injury. Threatened abrasions of the elbows and knees from rubbing may be averted by the use of icthyol collodion, a useful protective covering which cannot readily be torn off.

While a great number of drugs have been advocated in the treatment of chorea, the two which have chiefly been considered to have a specific influence over the disease are the *salicylates* and *arsenic.* The basis of salicylate therapy is the theory that chorea is usually a manifestation of the rheumatic fever infection. The reader should always remember, however, that the various statistical reports of clinical benefit from the use of any particular drug are very poor evidence of the specific value of that drug. It has not been definitely proved that salicylates have any etiotropic action in rheumatic fever. The only thing which has been proved is their influence in controlling the pain of the disease. In my opinion, there is little evidence that salicylate therapy has any specific influence in modifying the course of chorea, in spite of the favorable results reported by many observers. There is no objection to trying salicylate therapy in chorea, but its chief value will be found in cases accompanying an attack of rheumatic fever and characterized by pain. As to *arsenic,* the reports in favor of its specific action are open to similar objections. I have never been able to convince myself that arsenic had any specific action in controlling the disease. There is, however, no objection to trying a course of arsenic treatment in any case provided that all the much more important general hygienic and dietetic measures are properly carried out. The usual form of arsenic used in chorea is Fowler's Solution, and this is given in small doses of about 3 minims three times a day at first, and the quantity given is gradually increased up to about 10 minims three times a day, or until slight toxic symptoms are produced. Large amounts of arsenic must not be given for too long a period, and the urine should frequently be examined for evidences of renal irritation.

The drug treatment of chorea must be mainly symptomatic. I am accustomed to use no drugs at all in mild cases with the exception of iron as a tonic, but as stated above, either arsenic or salicylate may be tried according to the fancy of the physician. In severe cases sedatives are required. They must be given in doses sufficient to produce an effect. I generally begin with sodium bromide, and if this fails to control the severe nervous and mental symptoms, I try such sedatives as codein, chloral, veronal, or trional. If these measures fail, morphin given hypodermically may be used as a last resort.

Good results have been reported from the use of lumbar puncture in chorea, and I believe this should be tried in any exceptionally severe case. A new treatment, with autoserum, is discussed under Problems and Research. Other symptoms, such as circulatory weakness, or anemia, should be treated symptomatically.

PROBLEMS AND RESEARCH.—It is quite possible that when another edition of this book is written, chorea will have to be definitely classified among the infections. The tendency is decidedly that way. Present day research on chorea is being devoted especially to the following aspects of the subject: (1) the bacteriology of the disease; (2) the pathological anatomy of the central nervous system; (3) the collecting of special clinical signs pointing toward an organic lesion of the central nervous system.

Recent researches on the bacteriology of chorea have resulted in the repeated finding of a microörganism either in the blood or in the tissues of the central nervous system. On the other hand, there have been many negative findings even when the research was carried out by skilled investigators. The microörganism most frequently found is a coccus belonging in the general streptococcus group, and in general similar to the coccus originally described by Poynton and Payne. There are consequently a large number of observers who believe that Sydenham's chorea is essentially a manifestation of rheumatic fever and that it represents an organic disease of the brain which attacks all parts of the cortex equally and impartially. There is some difference of opinion among those who hold the view that Sydenham's chorea is essentially a manifestation of rheumatic fever infection, as to whether the disturbance in the central nervous system is caused by the direct presence of bacteria, or by the presence of bacterial toxins absorbed from some other part of the body. The finding of bacteria in certain cases supports one view, and the negative findings in other cases support the other. This is one of the questions which may be settled in the future. There are some other authorities who believe that chorea is always a manifestation of infection, that rheumatic fever is the commonest infection which produces the manifestations of chorea, but that other infections may at times produce the disease. This view would explain the cases which occasionally have been observed to follow other infections, such as scarlet fever. It is notable that most of the infectious diseases other than rheumatic fever, with which chorea has been associated, are conditions in which streptococci are frequently found as a secondary invader. The views of Rosenow on the transmutation of bacteria of the streptococcus-pneumococcus group, and on the ability of this group of organisms to change their selective affinities from time to time, is suggestive in connection with the occurrence of chorea, not only as a manifestation of rheumatic fever, but of other streptococcus infections.

Still another group of authorities, while admitting the frequent connection of chorea and rheumatic fever, believe that chorea represents a functional disturbance due to a great variety of causes. Among these causes infection is one, and of the infections rheumatic fever is the commonest, but they believe that the various factors of hygiene and environment which produce functional disturbance in general may be responsible for some cases of chorea without the medium of infection. This view would explain the cases in which chorea has apparently followed fright or other mental shock. The adherents of the infectious theory believe that such cases are too few in number to be significant.

The question of the pathological anatomy of chorea is closely connected with that of its bacteriology. It is obvious that research upon this aspect of the subject has been greatly hampered by the comparatively small number of fatal cases of this disease. Nevertheless, the view appears to be gaining ground that chorea does not represent merely a functional disturbance of the central nervous system, but that it is characterized by organic lesions of the brain which, while only temporary, are none the less actually present. There has been much interesting work upon this subject. Loubet in a very elaborate monograph (Thêse de Toulouse, 1912), concludes that the lesion is a mild encephalitis caused by the toxins absorbed from an infection without the actual presence of organisms in the tissues of the nervous system. The majority of French authorities appear to be inclined toward this view.

The difficulties attending the study of the lesions in chorea have stimulated the neurologists to a more thorough investigation of the clinical manifestations of the disease. They have been accumulating all sorts of signs brought out by special tests which point toward localized lesions in chorea. Thus, there is constantly accumulating additional evidences pointing toward the organic nature of the disease.

As far as treatment is concerned, it seems that in order to make further progress we must await further knowledge of the etiology and pathological anatomy of the disease. In the meantime, treatment must be mainly symptomatic.

A new treatment for chorea, now on trial, consists in the introduction into the spinal canal of some of the patient's own blood serum; after the withdrawal of a corresponding quantity of cerebrospinal fluid. According to the reports, the effect is very strikingly favorable in many severe cases, the incoördinate movements ceasing permanently within a few hours. Some cases are uninfluenced. This treatment at times is followed by temporary opisthotonos, and symptoms suggestive of anaphylactic shock. I have been informed that the favorable effect on the chorea is most marked in the cases in which the treatment produces the most marked disturbance. It is possible that the favorable effect of this treatment may be explained by the effect of suggestion on a chorea which has become a habit spasm.

SPASMODIC NEUROSES

Among the great variety of symptoms of purely functional disturbance of the nervous system seen in childhood, only comparatively few are sufficiently clear-cut and characteristic to warrant separate description. There are certain conditions accompanied by involuntary spasm of the voluntary muscles which apparently are pure neuroses, that is, which represent a purely functional disturbance of the central nervous system.

HABIT SPASM.—This term is used to describe a condition in which certain spasmodic muscular movements become habitual and entirely involuntary.

ETIOLOGY.—The causes are the general ones which produce purely functional disturbance of the nervous system. It is probable that in the very beginning the children make some movement voluntarily for some purpose which is clear to their own mind. The development of this movement into a habit spasm, however, is a pure neurosis. The patients usually have a neurotic family history. Their general health is below the normal. Such conditions as over-stimulation of the nervous system through excessive excitement or over-work at school, play an important part. Various factors in hygiene and environment may act as contributing causes. Sometimes there is a definite exciting cause, such as an acute illness. The condition when fully developed is really little more than an exaggerated nervousness which takes the form of the repetition of a certain movement until the fixed habit becomes established.

SYMPTOMS.—The spasmodic movement most commonly seen is in the muscles of the face, but almost any part of the body may be affected. The most common forms are a sudden frowning, blinking, or raising of the eyebrows, some special or peculiar grimace, or a sudden grinding of the teeth. In other cases there may be a sudden twitching of the head, shrugging of the shoulders, or jerking of the upper extremities. The lower extremities are very rarely involved. In some cases the respiratory muscles are affected, giving some form of sigh, sob, or cough.

In the beginning the spasmodic movements are not very frequent but as the habit becomes more and more firmly fixed, the spasms tend to recur more and more frequently, until in severe cases they may be almost continuous. In some cases there is a change in the form of the spasm during the progress of the disease.

DIAGNOSIS.—The condition is chiefly to be differentiated from chorea. Both chorea and habit spasm are usually described by parents as "nervousness" and the physician must clearly differentiate between the two conditions, as the prognosis and treatment are entirely different. In habit spasm the muscular contractions are limited to one part of the body, or to one group of muscles. In chorea, while there may be some irregularities in the distribution of the incoördinate movements, they are always more generally distributed than in habit spasm. The duration of the affection is much longer in habit spasm than in chorea. The differentiation is usually very easy if the frequency of habit spasm in early life is not forgotten.

PROGNOSIS.—This depends largely upon how soon treatment is begun. After the habit has existed a long time and has become firmly fixed, treatment is very unsatisfactory, and in many neglected cases the disease continues throughout adult life uninfluenced by treatment. On the other hand, when the condition occurs in early childhood, and is recognized at an early period, the prognosis is good.

TREATMENT.—All the conditions of the child's life and environment should be investigated, and every possible error should be remedied. Every measure which conduces toward the building up of the general health should be used. Any local cause which could act as a source of irritation to the nervous system should be removed. The rest of the treatment is moral and is carried on along the same lines as are used in the treatment of enuresis. Punishments are of no use and should be avoided, for in children of this age they only aggravate the condition; rewards are much more effective. Every effort should be made to win the child's confidence and to gain his intelligent coöperation. In this way children can be gradually taught to make a continuous voluntary effort to control the spasm, which eventually will result in success. In many cases, having the child sit before a mirror while making the effort to control the spasm, is very effective.

ROTARY AND NODDING SPASM OF THE HEAD.—These are neuroses which are seen chiefly in infancy.

ETIOLOGY.—The causes of these neuroses are exceedingly obscure. There appears to be very little relation to the general etiological factors in hygiene and environment which produce other neuroses. There is also no very definite relationship to a neurotic constitution. Some observers have attributed rotary spasm of the head to the fact that the infant was kept in a poorly lighted room, which compelled him to assume an unnatural position of the head in order to see the objects presented to his vision. Some cases have been cured by being placed in well-lighted rooms, but it is probable that this explanation is not applicable to all cases.

SYMPTOMS.—The disease is seen most often between the ages of three and eighteen months. The rotary spasm is the commoner. It consists of a peculiar side to side oscillation of the head, which may be either slow or rapid; in some cases it is almost continuous. Nodding spasm consists in a forward and back movement. At times, both forms are combined. Nystagmus is frequently an associated condition.

PROGNOSIS.—This is favorable, spontaneous recovery usually occurring in the course of a few months.

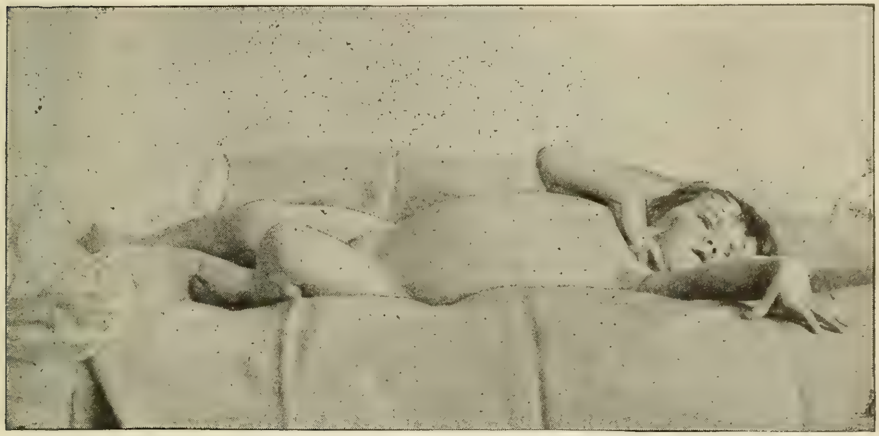
TREATMENT.—No other treatment is indicated than the making sure that the child is kept in a properly lighted room.

NYSTAGMUS.—This is a rhythmical, involuntary movement of the eyeball, usually involving both eyes. In the majority of cases, nystagmus is symptomatic of some other diseased condition. It is seen both in organic diseases of the eyes and in organic diseases of the central nervous system. It may be due to blindness, or any lesion of the eye which can produce that condition, or it may be due to errors in refraction. Nystagmus is a symptom of a great variety of organic diseases of the central nervous system in which it does not have very much diagnostic significance.

There are, however, a certain number of cases in which nystagmus appears to be a purely functional disturbance. In these cases the causes are probably those of the neuroses in general. The prognosis is favorable when nystagmus represents only a functional neurosis, and the treatment is that of the neuroses in general. The condition of the eyes and of the central nervous system should be investigated in every case, in order that the physician may not overlook an organic cause.

ATHETOSIS.—This name is used to describe a peculiar form of chronic spasm which usually involves the hand, but which in some cases may involve the foot, or even the face. In the majority of cases only one side is affected, but in some cases the movements are bilateral. The movement is slow. There is usually incoördination and sometimes a certain amount of muscular rigidity. The condition is most often seen as a symptom of an organic disease of the central nervous system, and is particularly common as a sequel of cerebral paralysis from hemorrhage or encephalitis. In these cases there is usually an associated hemiplegia. The same movements, however, have been observed in children who were apparently healthy, and in these cases it appeared to represent a pure neurosis of obscure cause. The prognosis for recovery is not very favorable. No special treatment is indicated.

FIG. 280



Congenital athetosis. Female, 2 years old

HICCOUGH.—This consists in spasm of the diaphragm, and is very common in early life. It is often seen in young infants, and is usually due to some reflex irritation originating in the stomach. In many cases, however, it appears to be a pure neurosis without any relation to the taking of food. In some cases it is caused by the swallowing of air. The treatment consists in the removal of any possible cause, and in the general hygienic treatment applicable to neurotic children. Any possible indigestion should be corrected. In infants the giving of a little sodium bicarbonate in hot water will often cause the expulsion of gas, and will at once relieve the symptom. In older children all faulty factors in hygiene and environment should be corrected. Children can be taught to arrest the spasm by the effort of holding the breath or of prolonged forced expiration.

RETARDED PSYCHICAL DEVELOPMENT

Arrested psychical development is a term used in speaking of an apparent lack of mental growth which is sometimes met with in infancy. So far as we know, it is a functional disturbance and not an organic disease of the brain. Infants with this affection develop both mentally and physically for a variable period, perhaps five or six months, and then continue to develop physically, but cease to develop mentally. This condition lasts for a variable period of months, when their mental development begins again, and, although for some time they are backward in comparison with other children of their age, they finally show no trace of an abnormal mental condition.

In some cases the arrest of cerebral development is associated with a condition of physical weakness, so that the power to sit and to walk at the usual age is delayed until the third or fourth year.

Arrested psychical development seems to be most commonly associated with repeated acute illnesses in infancy. Nothing more definite is known as to its etiology.

The differential diagnosis of this condition from defective mentality due to organic disease has been considered under Defective Cerebral Development. The prognosis is favorable, and no treatment is necessary.

RETARDED SPEECH

When during the second year the power of speech does not develop with the usual rapidity, it is spoken of as retarded speech.

The lack of power to speak may be due to a simple retarded but otherwise normal development of certain portions of the brain, or to organic or functional cerebral disturbance. It may also arise from abnormal conditions outside of the brain. The cases which are caused by a permanent organic lack of development may be of congenital origin, or may be due to an arrested cerebral development produced by a number of causes. These causes are various acquired lesions which produce serious interference with the cerebral growth and defective development of the bulbar nuclei. There is a temporary functional aphasia seen in a child who has already learned to talk. It is usually associated either with a fright or with an acute illness. A child may for a time during a severe illness, and after convalescence has been established, apparently be unable to use the words that it was accustomed to speak before the illness. Retarded speech may also be caused by such physical defects as disease of the ear, resulting in deafness, and from such a physical malformation of the mouth, palate, or vocal cords as to render articulation impossible. In this connection stammering may be spoken of as a cause of retarded speech.

When called upon to decide why a child is unable to speak, the previous history should be carefully investigated, and a thorough physical examination should be made. In this way organic disease of the brain can be eliminated by means of the absence of the usual symptoms of such disease, such as defective mentality, or spastic paralysis, and by ascertaining that the child has not had any cerebral disease sufficiently severe in its character to interfere with the development of the centers of speech. After determining that the child is not an idiot, the ears and the mouth should be examined. If the child is deaf, there is a good reason for his not being able to speak. Even when young children have learned to speak fairly well, if they later become deaf from a disease like scarlet fever, they are very apt to become mute also. When a lesion of the ear has occurred before the child has learned to speak, he almost invariably is found to be a deaf-mute, although there may be no defects in the mechanism of articulation or in his mental condition. It is seldom that any defect in the mouth or throat is found which interferes with articulation, except in cases in which very extensive lesions are present, such as cleft-palate, and sometimes enlarged tonsils combined with a high-arched palate and a large adenoid growth. The tongue-tie which the parents usually consider to be the cause of the retarded speech is seldom present. When no symptoms of organic, functional or developmental cerebral disease exists, when there is no physical deformity, and when the child hears well and seems bright and well developed in other ways, the parents, as a rule, may be assured that the speech is merely retarded and will probably develop later.

Stuttering, stammering and lisping are functional disturbances which do not require detailed description. The treatment of severe cases should be placed in the hands of a specialist in the use of vocal gymnastics.

HEADACHE

When pain in the head occurs in early life it is to be regarded more seriously than at a later period, as it is more apt to indicate some grave central lesion. The various forms of headache due to organic disease which arise in children can be spoken of best as symptomatic of the various diseases in which they occur, such as typhoid fever, and tumors of the brain.

There also appears to be a type of headache which occurs in the later years of childhood irrespective of any definite disease and is often unaccompanied by nausea. These headaches, as a rule, are not of serious import, and are usually classed under the term functional. They occur irregularly, and may be in any part of the head. They are often so severe that the child has to lie down. The intervals between the attacks are variable, and the length of the attacks varies from two to three hours to a day. Of these functional headaches the most frequent form in children is that due to anemia. It is always, however, wise to look upon this form of headache as a possible symptom of some undetermined disease.

Although in many cases headaches are caused by an improper regulation of the diet, yet there is evidently some other cause which we do not recognize in their production, as with exactly the same diet for many months a child will show no symptoms whatever of headache. In like manner, although we know that headaches in children may be due to constipation, yet this class of cases occurs whether constipation is present or not. The source of the headache cannot be determined by the part of the head affected.

Migraine also may exist in children, and is characterized by severe pain in the head, sometimes unilateral, sometimes bilateral, accompanied by nausea, dizziness, and generally vomiting. The attacks occur at irregular intervals, and usually last the greater part of a day. They may be brought on by apparently slight causes, such as over-fatigue, eye-strain, or very mild indiscretions of diet, in those predisposed to them. These headaches are markedly hereditary. Migraine may appear in later life in cases which in childhood have been subject to persistent or periodic vomiting.

Although all these forms of headache are ordinarily very intractable to cure, especially when no bad hygienic surroundings exist which might account for them, and when the child does not lead a sedentary life, yet, as a rule, the attacks have a tendency to lessen and disappear as the child grows older.

One of the most common causes of headache in children is strain of the eyes. In all cases of headache in children the cause of which is not evident, a careful examination of the eyes should be made, even though there be no symptoms which point to the eyes themselves.

TREATMENT.—The treatment of headache should be directed to that of the disease which is causing the disturbance when it can be detected, as in anemia or hyperemia from various causes, gastric disturbances, the prodromal stages of the acute infectious diseases, malaria, uremia, and other toxic causes; when, however, no cause can be found, a darkened room and sufficient bromide of soda to allay pain and produce sleep are indicated. Antipyrin, phenacetin, ammonol, and other analgesics may be employed temporarily to relieve the headache while the cause is being sought and treated.

NEURALGIA

Neuralgia is a functional affection of the sensory fibers of the peripheral nerves, represented by pain. The causes are probably multiple, including the possibilities of infection, autointoxication, and errors in hygiene and environment. It is rare in infancy and early childhood. It may occur in very different localities, and may be represented by intercostal neuralgia or by various mild forms of flitting pain in different parts of the body. Neuralgia of the trifacial nerve is the most severe form observed in children, the most frequent cause of which is chronic disease of the nasopharyngeal space and of the antrum of Highmore, especially after influenza.

Sciatica is a form of neuralgia which may occur in children, and is characterized by pain and tenderness along the course of the sciatic nerve, especially at its point of exit from the pelvis.

In most cases of neuralgia temporary relief from the pain can be obtained by the use of aspirin. Every effort should be made to discover a possible cause in the hygiene and environment of the child.

DISTURBED SLEEP

Disturbances of sleep in childhood may be due to organic disease of the central nervous system, but the majority of cases represent a purely functional disturbance. A certain number of cases are symptomatic of recognized diseased processes elsewhere. Other cases represent the direct effect upon the nervous system of a great variety of the etiological factors which are concerned in the production of functional disturbance. It is only this latter group of cases which are strictly to be classified among the primary functional disturbances of the nervous system, but for the sake of completeness I shall enumerate the principal causes of disturbed or restless sleep whether the cause be disease elsewhere or not.

ETIOLOGY.—Among the disease processes in various parts of the body which may cause more or less disturbance of the sleep are the following: Indigestion; hunger, or relative starvation; otitis media; dentition; acute infectious diseases; obstructed respiration from adenoids or enlarged tonsils; nocturnal attacks of asthma; hip-joint disease; organic cardiac disease; pulmonary diseases accompanied by dyspnea or cough. Closely connected with these diseased conditions are certain conditions which may produce simply discomfort on the part of the child, such as lack of fresh air in the sleeping room, excessive or insufficient bed clothing, cold feet, wet diapers, and so forth.

When disturbance of sleep represents a true functional disturbance of the nervous system, the cause is most frequently some violation of the hygiene of the nervous system. The most common cause is excessive over-stimulation from over-pressure in schools, or from the telling of exciting stories, or from indulging in violent and exciting play toward bedtime. All such violations of the hygiene of the nervous system result in a condition of general nervous exhaustion, of which disturbance of sleep is a very common symptom. Sometimes the disturbance is the result of a bad habit, such as rocking a child to sleep, giving it night feedings, and allowing a child to go to sleep in a lighted room.

SYMPTOMS.—Real insomnia is comparatively rare in early life, although it may be produced by the same causes. The usual condition is one of restlessness, in which the child during sleep is disturbed and restless, waking many times during the night. Many children wake up with a scream and immediately fall asleep again. The conditions of disturbed sleep which are due to violations of the hygiene of the nervous system are closely connected with the disease pavor nocturnus, which will be described below.

TREATMENT.—The essential treatment of sleeplessness in early life consists in removal of the cause. This will involve a careful search for all the possible causes enumerated under etiology. In cases which are due to distinct diseased conditions, such as indigestion, the treatment is that of the underlying cause and is mainly dietetic. In the cases in which the disturbance of sleep is due to a condition of nervous exhaustion produced by faulty hygiene of the nervous system, all faulty conditions should be immediately remedied. In many cases a change of nurses is very valuable in the treatment. Treatment of any general conditions of anemia and malnutrition which may be present, should be instituted.

Under almost no circumstances in early life should the physician ever countenance the use of drugs to promote sleep. The only exception is when sleeplessness is due to severe acute disease. In all other conditions drug treatment should be avoided, no matter how severe the insomnia is. When sleeplessness is symptomatic of acute disease, the least harmful and the most effective drug in early life is sodium bromide. In resistant cases trional may be tried, but the opiates should never be used unless the sleeplessness is entirely due to severe pain.

In the majority of cases in children not suffering from acute disease, a quiet darkened room, with plenty of fresh air, the stopping of eating and drinking during the night, and the correction of any manifest errors in hygiene will speedily suffice to bring about a cure. A warm bath at bedtime will be found useful in many cases.

PAVOR NOCTURNUS

(Night Terrors)

This is one of the commonest manifestations of functional disturbance of the nervous system in early life. The essential feature of the condition is the sudden waking of the child in a state of nightmare or fright.

ETIOLOGY.—While the condition is more common in children who have a constitutional neurotic tendency, the chief cause is persistent violation of the hygiene of the nervous system. The condition is seen most frequently in children whose nervous systems are habitually over-stimulated. A very common cause is over-pressure at school, and pavor nocturnus is not uncommon in children who are striving to excel in their studies and to get good marks, and who hold the position of " teachers' pets." Other causes are too much excitement in the home life. The frequent taking of children to theatres or moving picture shows, and all forms of unusual or exciting amusements are common causes of this condition. I have seen a very severe case develop in a young child whose mother took it for a ride on a roller coaster. Fright is a frequent cause. The telling of exciting or horrible stories, especially toward bedtime, is another frequent violation of the hygiene of the nervous system which leads to pavor nocturnus. In general it may be said that the disease may follow all forms of mental over-stimulation. There are certain other contributing or precipitating causes which are found associated with certain cases. I have noticed that pavor nocturnus is particularly common in children who eat too much sweets, or in whose diet there is an excessive balance of carbohydrate. It is well always to look for other symptoms of chronic carbohydrate indigestion in these cases. It is possible that other forms of indigestion may act as the precipitating cause of an attack. In some cases the precipitating cause may be partial asphyxia from adenoid tissue in the pharynx.

SYMPTOMS.—The general symptoms of disturbed sleep described above are often present, and the attack may be only the culmination of such disturbance. In mild cases the child wakes up in a state of fright and excitement. He often says he has had a bad dream, or talks of objects which he has seen in his dream. His mind, however, is clear, and he recognizes those about him. It may be a long time before the child becomes sufficiently calm to go to sleep again. In severe cases the attack comes on more suddenly and often is not preceded by restlessness. The child suddenly sits up in bed in a state of apparently extreme terror, with his eyes wide open and staring. He does not recognize those about him, nor appear to have any consciousness of his surroundings, but his mind appears to be entirely taken up with some hallucination which has produced the fright. He often mentions, and sometimes even points toward the objects which figure in his hallucination, bears, dogs, and black cats being the visions which I have heard referred to most. In these severe attacks the child does not come to full consciousness and soon goes to sleep again. On the following day there is no recollection of what has occurred, whereas in the mild cases the child often remembers having had a bad dream.

Some writers believe that the mild and severe forms represent different disturbances, but in my opinion, they are the same in etiology, differing only in severity. I have frequently seen mild cases gradually assume a more severe type. The attacks vary very much in frequency, sometimes coming every few nights, and sometimes occurring only at intervals of several months. It is a curious fact that in the severe cases the hallucination in the successive attacks is very apt to be the same, which suggests that the original cause was some fright connected with that particular object. I have not, however, been very successful in eliciting a history of a fright of this kind in cases of pavor noctrunus. I have, however, one case on record in which a child looking out of a window saw a horse fall in the street and bleed to death. This patient developed a typical case of the severe form of pavor nocturnus, the hallucination being always the bleeding horse.

DIAGNOSIS.—The diagnosis in cases of pavor nocturnus is simple. The mother's description is often misleading, suggesting the possibility of an epileptic attack. Sometimes it is quite difficult to obtain from her an adequate description. In such cases it is sometimes a good plan for the physician to depart from the usual rule of letting the mother tell her story without prompting, and to describe a typical attack of pavor nocturnus, asking her, " does the child act like that? " The attacks are really so typical that the mother often recognizes at once the exactness of the physician's description, when she cannot describe the attacks herself.

PROGNOSIS—The prognosis is favorable. Some writers believe that the severe form is often a precursor of more serious disturbances, such as epilepsy or insanity, but in my experience, even the severe forms quickly yield to proper treatment.

TREATMENT.—The treatment consists in a most careful regulation of the hygiene of the nervous system in accordance with the principles described in Volume I, page 46. It is particularly important to remove every possible cause of mental over-stimulation. The diet and all faulty conditions in hygiene and environment should receive attention. I usually exclude cane sugar from the diet, and often reduce the proportionate amount of carbohydrate. It is often advisable in beginning treatment to have some person in whom the child has complete confidence sleep in the same room. Sometimes a change of nurse has a good effect; sometimes a complete change of environment may be necessary. If the attacks are very frequent, a dose of bromide at night may be given for a short time after treatment has been begun, until sufficient time has elapsed for the general treatment to take effect.

V. INFECTIONS

ACUTE MENINGITIS

Acute meningitis is the commonest infection of the central nervous system in early life. The disease in all its etiological forms is very much more common in children than in adults. Classification of the various forms of acute meningitis upon an etiological basis has been made possible by the general adoption of diagnostic lumbar puncture, which gives distinct evidence as to the particular micro-örganism which in each case is the cause of the disease. The micro-örganisms concerned in the production of meningitis are in the order of frequency in this locality the following:

1. The bacillus tuberculosis
2. The diplococcus intracellularis
3. The pneumococcus
4. The streptococcus pyogenes
5. The bacillus influenzae
6. The staphylococcus pyogenes

A few cases have been reported of meningitis due to the typhoid bacillus, the gonococcus, the colon bacillus, and other organisms. These cases, however, are too rare to require special description. The order of frequency given above is that which prevails in times when there is no especial prevalence of epidemic cerebrospinal meningitis. Even in a time of epidemic, it is questionable whether the number of cases of the epidemic form equals that of the tuberculous form, although the number is proportionately very much greater. The prevalence of the epidemic form varies very much in different localities. There are many parts of the country in which sporadic cases are seldom or never seen, and in which epidemics are rare. In the vicinity of Boston, however, a certain number of sporadic cases of the epidemic form are reported each year. The epidemic form has on account of its epidemic occurrence and specific therapy been described in the division on the specific infectious diseases. Tuberculous meningitis is sufficiently characteristic in its pathology and symptoms to require description under a separate heading. The forms which will here be considered under the title of acute meningitis are those due to the pneumococcus, the streptococcus, the influenza bacillus, and the staphylococcus.

PNEUMOCOCCUS MENINGITIS

This is the commonest of the forms included under this heading which is seen in early life.

ETIOLOGY.—This disease is due to invasion of the meninges by the pneumococcus of Fränkel. The disease is probably always secondary. In the great majority of cases pneumococcus meningitis occurs as a complication of some form of pneumococcus infection of the lungs. There are usually signs of pulmonary consolidation, and the meningeal symptoms are usually preceded by the clinical picture of some form of pneumonia. The route of invasion of the meninges is undoubtedly through the general circulation. Cultures from the heart's blood made post-mortem, and blood cultures taken during life in pneumococcus meningitis, almost invariably reveal the presence of the pneumococcus in the blood. Consequently pneumococcus meningitis represents one of the results of the generalized pneumococcus septicemia, which so frequently accompanies severe cases of lobar pneumonia and other forms of pneumococcus infection of the lungs. In some cases, in addition to pneumonia, such complication as empyema, pericarditis or peritonitis have also been present. Some cases occur in which the pneumococcus meningitis is apparently primary, no signs of consolidation nor other evidences of pulmonary infection being found. It is probable that in these cases the meningitis is secondary either to a latent pneumonia, or possibly to some other focus of pneumococcus infection, such as a tonsillitis or otitis media.

Pneumococcus meningitis is a comparatively late complication in lobar pneumonia. The meningeal symptoms which are so often seen at the onset or in the early stages of pneumonia do not generally represent a true infection of the meninges, but rather an irritation from the toxins absorbed from the lungs. At times the disease develops quite late during the convalescence from an attack of pneumonia, but in the majority of cases it develops between the fifth and the tenth days.

This form of meningitis is more common in infants than in older children, and is frequently seen in very young infants.

PATHOLOGICAL ANATOMY.—The lesions produced by pneumococcus infection of the meninges do not differ in any important respect from those of septic infection with any of the pus producing microörganisms. The inflammatory process is characterized by purulent, sero-purulent, and fibrino-purulent exudation, chiefly in the sulci along the vessels, or in the choroid plexus, and is usually most pronounced at the base, but at times is more marked on the convexity of the brain, especially over the parietal and occipital lobes. The meninges of the cerebellum are always affected. The meninges of the entire brain are rarely involved, although occasionally large areas are included in the inflammatory process.

The inflammation is confined chiefly to the piarachnoid. The dura is but slightly involved, and the subdural space is increased by the fluid exudate.

In the most acute cases, in which the macroscopic changes are very slight, the lesions are shown by microscopic examination to consist chiefly of purulent infiltration of the meninges, with injection of the blood vessels. The polynuclear leukocytes are massed in some places, scattered in others, and with but little fibrin between them. The absence of eosinophilic cells is notable. There is no evidence of proliferation of tissue.

In the more advanced cases, on the other hand, there are large numbers of cells, chiefly polynuclear leukocytes, lying in the dilated lymph-spaces of the tissue. The presence of red blood-corpuscles is rare. Fibrin is present in greater amount in this form than in any other variety of acute meningeal infection. Proliferative changes within the intima of the arteries are common in pneumococcus meningitis, but rare in the epidemic form. The cord is involved much less than in the epidemic form. There is also usually less distention of the ventricles. The cerebrospinal fluid is cloudy, increased in amount, and contains numerous pus cells and fibrin flocculi.

SYMPTOMS.—In cases which are apparently primary, the symptoms are indistinguishable from those of an epidemic meningitis, and indeed the clinical description of that disease given in Volume I, page 623, will apply to any form of acute meningitis. When pneumococcus meningitis occurs as part of the terminal infection in a general pneumococcus septicemia, it may be latent and only found at autopsy. In the majority of cases, however, the meningeal complication in the course of an attack of pneumonia is indicated by a very abrupt onset of vomiting or convulsions, followed by delirium or stupor. The temperature is usually markedly elevated. Rigidity of the neck is usually present, but is a less constant sign than in epidemic meningitis. Opisthotonos is comparatively rare. Abnormalities of the pupils are less common than in the epidemic form, and photophobia is often not marked. The tonic spasm of the muscles of the extremities, which is so common a symptom in the epidemic form, is usually less marked in pneumococcus meningitis, and may be absent. Kernig's sign, Brudzinski's neck sign, and abnormalities of the reflexes are also somewhat less common than in the epidemic form. In general it may be said that in pneumococcus meningitis the signs of local irritation of the nerve tissues are apt to be somewhat less marked than in the epidemic form, the patient appearing rather to be overwhelmed with an intense toxemia. Unconsciousness often does not develop so early in the disease as in severe cases of the epidemic type, but is sure to develop sooner or later.

After the full development of the symptoms in pneumococcus meningitis, the course of the disease is very short, shorter indeed than in any other form of meningitis. Death usually occurs within three or four days of the first symptom. Occasionally, however, very prolonged cases are seen which in their general course bear a close resemblance to epidemic meningitis.

DIAGNOSIS.—A very detailed clinical description of pneumococcus meningitis is unnecessary, because the diagnosis does not depend mainly upon the symptoms. The physician must be able to recognize meningeal symptoms, but there is no symptom or combination of symptoms by means of which a positive diagnosis of meningitis can be made, or by means of which one can arrive at a differential diagnosis between the various forms of meningeal infection. The diagnosis can only be made by lumbar puncture. The indications for lumbar puncture in cases of suspected meningitis have been described in Volume I, page 631. When meningeal symptoms occur at the onset of a case of lobar pneumonia, the probabilities are so strongly against a true infection of the meninges, and so strongly in favor of the meningeal symptoms being due to toxic irritation, that lumbar puncture may be postponed. When, however, in the course of a case of pneumonia, meningeal symptoms suddenly develop at some later period, lumbar puncture should always be performed.

The gross appearance of the cerebrospinal fluid obtained by lumbar puncture in pneumococcus meningitis does not differ from that obtained in cases due to the meningococcus. The fluid is usually under increased pressure and presents a greater or less degree of turbidity. Microscopic examination shows that the cells are chiefly of the polymorphonuclear variety. Pneumococci are ordinarily very abundant and very easily found in cover glass preparations. In fact, microörganisms are usually more numerous in the cerebrospinal fluid in the pneumococcus form than in any other form of acute meningitis. The pneumococcus is recognized by the fact that it usually occurs in pairs, but sometimes in short chains; in the cerebrospinal fluid the chains are rarely more than four cells in length. The cells composing the pairs lie end to end with reference to their long axis. The organism is gram positive. The pneumococci grow very readily in cultures from the cerebrospinal fluid.

PROGNOSIS.—The prognosis is bad. There are a few recorded instances of recovery from pneumococcus meningitis in adults, but as far as I know, none in children.

TREATMENT.—Future hope in this disease lies in the advance of serum therapy. The present situation with regard to antipneumococcic serum has been discussed under Lobar Pneumonia. There are as yet no recorded cases of recovery from pneumococcus meningitis under the use of antipneumococcic serum, although the cases in which it has been tried are comparatively few. The chances are rather against effective serum therapy at present, on account of the multiplicity of the strains of pneumococci, each with its specific immune reaction. Nevertheless, I believe that in all cases of pneumococcus meningitis in which the diagnosis has been properly confirmed by lumbar puncture, antipneumococcic serum should be tried. On the ground of the analogy of epidemic meningitis, it would seem useless to give the serum by the subcutaneous or intravenous routes. It should be injected into the spinal canal. The doses should be about the same as those of the antimeningitis serum used in the epidemic form.

Apart from serum therapy the treatment must be purely symptomatic. It resembles that of epidemic meningitis described in Volume I, page 642.

STREPTOCOCCUS MENINGITIS

ETIOLOGY.—This form is due to infection of the meninges with the streptococcus pyogenes (streptococcus hemolyticus). The infection of the meninges is always secondary. In the newborn, it occurs as part of a general septic infection which usually originates from the umbilicus. It is the usual etiological form which follows infection of a spina bifida. It may follow otitis media with mastoiditis or spetic sinus thrombosis. It is the usual form of meningitis which sometimes follows injuries to the head or operation upon the brain. It is sometimes seen as a complication of other infectious conditions about the cranium, such as erysipelas of the scalp. While streptococcus meningitis is a comparatively rare complication of otitis media, I have seen more cases in which it was due to that cause than to any other.

PATHOLOGICAL ANATOMY.—The lesion is essentially a widespread purulent inflammation of the pia mater, with a very abundant exudate of pus but with less fibrin than in either the epidemic or the pneumococcus forms. Hydrocephalus is very apt to develop very early in this form of meningitis, and is usually to be found at autopsy.

SYMPTOMS.—In general the symptoms of streptococcus meningitis are not distinctive and resemble those of any form of acute meningitis. I have noticed, however, certain differences which, while by no means constant or characteristic, are sometimes so suggestive as to enable the physician to make a guess that he is dealing with the streptococcus type. The onset is usually less sudden than in either the epidemic or the pneumococcus forms. In some cases, while not sudden, it is rapid; in other cases it is so gradual as to suggest the possibility of tuberculous meningitis. The initial symptoms are usually vomiting and headache. The temperature from the beginning is apt to be extremely high, hyperpyrexia being commoner in this type than in any other. The various meningeal signs, such as rigidity of the neck, Kernig's sign, Brudzinski's sign, and so forth, are often not present at the very beginning of the disease but inevitably develop during its course. Delirium and unconsciousness are often not marked at the beginning, but develop more gradually. Convulsions are rather more frequent than in either the epidemic or pneumococcus types. In general the course of the disease is apt to be notably slower than in the pneumococcus cases, or in epidemic cases of the same general severity. There is a steady progress toward a fatal ending.

DIAGNOSIS.—The diagnosis depends upon the results of lumbar puncture. The cerebrospinal fluid obtained is usually markedly purulent. The cells are of the polymorphonuclear variety. The microörganisms are usually to be found in cover glass preparations and grow readily in cultures. The recognition of the streptococcus in the cerebrospinal fluid is always easy. It almost invariably forms long curved chains, which are unmistakable. It is Gram positive.

PROGNOSIS.—This is absolutely unfavorable. There are on record no authentic cases of recovery from streptococcus meningitis.

TREATMENT.—The only possibility in specific therapy is the injection of an antistreptococcic serum into the spinal canal. While in the present stage of serum therapy this procedure does not offer very much prospect of success, in view of the fatality of the disease I believe it should be tried in all cases in which the diagnosis is properly substantiated. Vaccine therapy obviously does not offer any prospect of success in so acute a process. The general treatment is symptomatic and has been described under Epidemic Meningitis.

INFLUENZA MENINGITIS

This form is not so very uncommon even when an epidemic of influenza is not prevalent. It is decidedly commoner in infants than in older children, and is seen with comparative frequency in the first year of life.

ETIOLOGY.—The microörganism is the bacillus of influenza (Pfeiffer's bacillus). It is probable that the infection of the meninges is always secondary to an influenza infection of the air passages, but in some cases the symptoms of the primary disease are not prominent and only the meningeal symptoms attract attention. The bacilli probably gain the meninges through the blood stream rather than through the cribriform plate, as has been suggested by some authorities. Blood cultures are usually positive.

PATHOLOGICAL ANATOMY.—The lesions show no essential differences from those of other forms of acute septic meningitis. In the cases which I have seen at autopsy, the exudate was usually purulent rather than fibrinous, and resembled that seen in streptococcus meningitis.

SYMPTOMS.—The symptoms very closely resemble those of epidemic cerebrospinal meningitis. The onset, however, is less rapid, rather resembling that seen in the streptococcus form. The usual symptoms of onset are headache, vomiting and fever; the temperature may be either moderate or high. Rigidity of the neck, Kernig's sign, and other meningeal symptoms are often not present at the very beginning, but soon develop. Unconsciousness does not occur so early as in the epidemic and pneumococcus forms, but develops sooner or later. The course is rather variable in length, in general being somewhat slower than that of either the epidemic or pneumococcus form, and rather resembles that seen in the streptococcus form.

DIAGNOSIS.—This depends entirely upon the results of lumbar puncture. The fluid in gross appearance does not differ from that seen in other forms, being turbid, and under increased pressure. On microscopic examination, it is seen that the cells are of the polymorphonuclear variety. In cover glass preparations made from the fluid the organisms are usually less numerous than in either pneumococcus or streptococcus meningitis. Their recognition by any one not accustomed to frequent examination of the cerebrospinal fluid is apt to be rather more difficult than in the other forms. The picture presented in cover glass preparations is often somewhat confusing, the general impression being that of a mixed infection of diplococci and bacilli. This is due to the fact that the bipolar staining of the influenza bacillus gives to that organism in stained cover glass preparations the appearance of a diplococcus. In ordinary stains with Loeffler's alkaline methylene-blue, this apparent diplococcus, although somewhat smaller, may suggest epidemic meningitis. In addition to these forms which resemble a minute diplococcus, bacillary forms are usually present. These bacillary forms present a great variation in length, and often quite long bacilli are seen which in no way resemble the forms which suggest a minute diplococcus. These long bacilli represent involution forms of the bacillus of influenza, and their presence while at first confusing is really a great aid in the diagnosis, if the frequency of their occurrence be borne in mind. Of course, in some cases the long bacillary forms are absent, but I have seen them in the majority of instances. Whenever the cerebrospinal fluid shows an apparent mixed infection with a minute diplococcus and a bacillus of varying length, the physician is justified in making a diagnosis of influenza meningitis. The organism is Gram negative, and is thus easily distinguished from the pneumococcus. Final proof of its identity can only be obtained by demonstrating that it will only grow on media containing hemoglobin.

PROGNOSIS.—The prognosis is very unfavorable. There have been a few recorded instances of recovery from influenza meningitis in early life, but such cases are extremely rare. All the cases which I have seen have progressed steadily to a fatal ending. The fact that so many cases occur in early infancy possibly partly accounts for the extreme fatality of this form of meningitis.

TREATMENT.—Intraspinal injections of anti-influenza serum should be given, the general plan of treatment being the same as that described for the use of antimeningitis serum in the epidemic form. Researches at the Rockefeller Institute have demonstrated that an animal serum can be produced, which in experimental infections in animals has a marked protective influence against influenza meningitis. This suggests that there is hope of obtaining a favorable result in man by means of serum therapy. The recorded cases of influenza meningitis in which serum therapy was used are still too few to allow any judgment as to the value of the treatment. One obstacle is that the diagnosis of this form of infection is rarely made in the early stages of the disease. Considerable time elapses before the examination of the cerebrospinal fluid is complete. It is only very rarely that serum injections have been given in the first few days. I have used serum therapy in influenza meningitis in eleven cases, all of which have been fatal, but in no case did I see the patient early enough to expect the best result from the treatment. Besides serum therapy, the treatment is wholly symptomatic.

STAPHYLOCOCCUS MENINGITIS

This form is quite rare in early life.

ETIOLOGY—The organism is the staphylococcus aureus or albus. Infection of the meninges is always secondary and follows the same conditions which produce streptococcus meningitis.

PATHOLOGICAL ANATOMY.—The lesion is a purulent inflammation which does not differ from that produced by the streptococcus.

SYMPTOMS.—The symptoms are not distinctive. In the few recorded cases the course has been somewhat more prolonged than in other forms.

DIAGNOSIS.—This depends upon the results of lumbar puncture. The staphylococcus is easily recognized in cover glass preparations from the cerebrospinal fluid.

TREATMENT.—Vaccine therapy should be employed in staphylococcus meningitis. There is more evidence of the value of vaccines as a weapon against staphylococcus infection than against any other form of infection. Not only that, but there are on record instances of recovery from staphylococcus meningitis under vaccine therapy. In these cases a visible improvement has followed each injection of the vaccine. An homologous vaccine should be employed whenever the physician is master of the technic of its preparation. Otherwise a stock vaccine may be tried. The initial dose should be 50,000,000, and the doses should be gradually increased 50,000,000 at a time until a response is observed. The intervals between the injections should be from two to three days. The rest of the treatment is purely symptomatic.

The following table shows the frequency of occurrence of the various characteristic symptoms and signs of meningitis in the most common etiological forms. It is based on a series of 142 cases.

TABLE 74

Characteristic Symptoms and Signs of Meningitis in the Various Forms

SYMPTOMS	TUBERCULOUS	EPIDEMIC	PNEUMOCOCCUS	STREPTOCOCCUS	INFLUENZA
Onset—					
Sudden	9	47	11	9	2
Medium	13	10	1	4	1
Gradual	37	3	0	2	1
Temperature—					
High	11	41	12	6	1
Moderate	18	17	0	0	3
Low	19	2	0	0	0
Cloudy mentality or delirium—					
Early	2	50	8	0	0
Developed	52	9	4	6	3
Late	4	0	0	0	0
Absent	0	1	0	0	0
Unconsciousness—					
Early	2	27	8	0	0
Medium	15	0	4	4	3
Late	41	0	0	2	1
Absent	0	24	0	0	0
Headache—					
Present	17	32	8	6	4
Doubtful	18	10	4	0	0
Absent	25	18	0	0	0
Rigid neck—					
Early	7	51	10	1	1
Developed	29	8	2	5	3
Absent	23	1	0	0	0
Kernig's sign—					
Early	7	35	11	0	1
Developed	21	5	1	6	2
Absent	31	0	0	0	1
Knee-jerks—					
Exaggerated	15	13	5	3	1
Normal	20	8	0	0	1
Absent	24	39	7	3	2

TABLE 74—*Continued*

SYMPTOMS	TUBERCULOSIS	EPIDEMIC	PNEUMOCOCCUS	STREPTOCOCCUS	INFLUENZA
Localizing neurologic signs—					
Early	2	4	0	0	0
Developed	19	6	1	4	2
Absent	39	50	11	2	2
Babinski's sign—					
Present	19	19	3	0	0
Absent	35	41	9	6	4
Convulsions—					
Early	8	8	2	0	0
Developed	12	1	0	5	1
Absent	40	51	10	1	3
Vomiting—					
Present	41	38	3	6	4
Absent	19	22	9	0	0
Pulse—					
Slow	15	9	0	3	0
Rapid	43	51	12	3	4
Cheyne-Stokes breathing—					
Present	18	8	1	2	0
Absent	42	52	11	4	4
Petechial eruption—					
Present	0	11	2	0	0
Absent	60	49	10	6	4

Table 74 shows the diagnostic characteristics of the cerebrospinal fluid in the active stages of the various forms of meningitis.

TABLE 75

Diagnostic Characteristics of the Cerebrospinal Fluid in the Active Stages of the Various Forms of Cerebrospinal Meningitis

	APPEARANCE	CELL COUNT	PREDOMINANT CELL	MICRO-ÖRGANISM
Normal fluid	Clear	Normal	Mononuclear lymphocyte	None found
Tubercular meningitis	Clear or slightly cloudy	Increased	Mononuclear lymphocyte	Tubercle bacillus in some (Acid-fast)
Epidemic meningitis	Cloudy	Increased	Polymorphonuclear neutrophil	Diplococcus intracellularis (Gram-negative)
Pneumococcus meningitis	Cloudy	Increased	Polymorphonuclear neutrophil	Pneumococcus (Gram-positive)
Streptococcus meningitis	Cloudy	Increased	Polymorphonuclear neutrophil	Streptococcus (Gram-positive)
Influenza meningitis	Cloudy	Increased	Polymorphonuclear neutrophil	Influenza bacillus (Gram-negative)
Staphylococcus meningitis	Cloudy	Increased	Polymorphonuclear neutrophil	Staphylococcus (Gram-positive)

TUBERCULOUS MENINGITIS

Tuberculous meningitis is a tuberculous infection resulting in an inflammation of the pia mater. It is by far the most frequent form of meningitis encountered in early life. Even when the epidemic form is extremely prevalent, the number of cases does not reach that of the tuberculous form which is seen at all times.

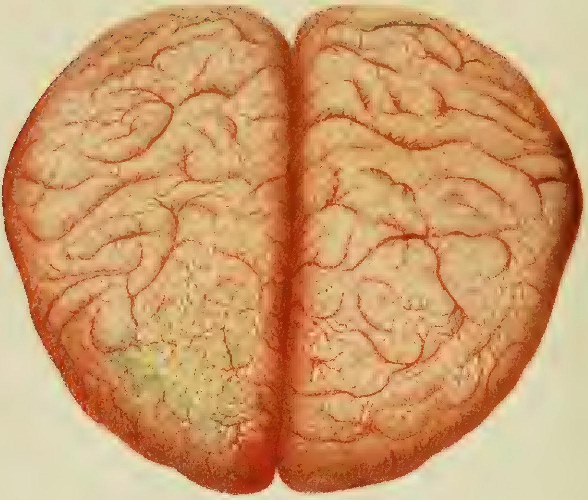
ETIOLOGY.—The pathogenesis of tuberculous meningitis has been discussed in the general article on Tuberculosis in Volume I. The occurrence of this lesion in a case of tuberculosis represents a low degree of resistance on the part of the patient. Of the various secondary manifestations of tuberculous infection in early life, meningitis represents a form to which infants and young children are particularly liable. The bacilli are carried to the meninges from the primary tuberculous lesions through the medium of the blood stream. In the majority of cases the meningitis represents only a part of a general miliary tuberculosis, and in these cases there are numbers of tubercle bacilli in the blood which are simultaneously deposited in the tissues of the pia mater. My own researches on this disease have suggested that there is another form of tuberculous meningitis which shows not only anatomical but even clinical differences from the ordinary form. In these cases the anatomical evidence suggests that only very few tubercle bacilli are originally carried to the meninges, and that they there form one or more localized tubercles which act as distributing foci, from which the infection gradually extends over the rest of the meningeal surface.

It should never be forgotten that tuberculous meningitis never represents a primary manifestation of tuberculous infection. It is always secondary to primary lesions which in the order of frequency are situated in the lungs, in the abdomen, or in the cervical lymph-nodes.

Tuberculous meningitis is most often seen in the first two years of life. It is commonest in the first year, but almost as common in the second year. After the second year its frequency is much less, and steadily diminishes throughout childhood. The frequency of this disease as a secondary manifestation of tuberculosis in infancy can only be accounted for by a lessened resistance toward a generalized spreading of the tuberculous infection at this age.

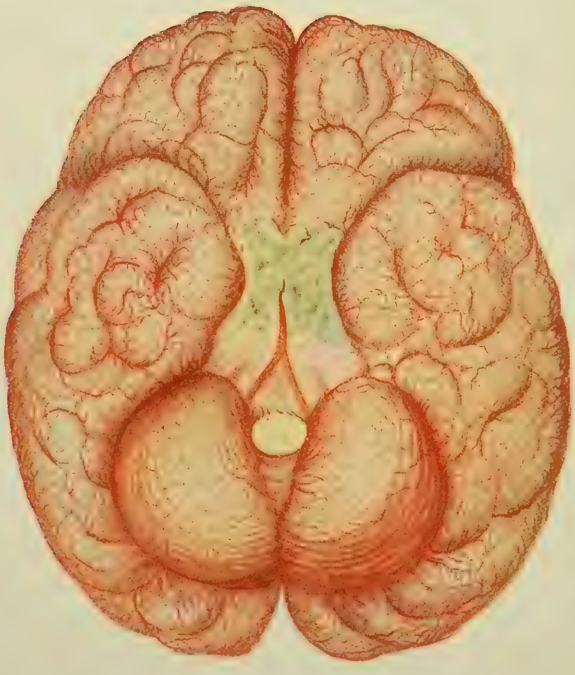
PATHOLOGICAL ANATOMY.—A knowledge of the general pathological anatomy of tuberculous meningitis is of great practical importance in acquiring a clear picture of the disease. The macroscopic pathological condition which is seen in the brain as a result of the action of the tubercle bacillus is a growth of miliary tubercles in the meninges. This growth is especially marked in the meshes

PLATE I*a*



Tubercular meningitis

PLATE I*b*.



Tubercular meningitis

of the pia mater along the course of the blood vessels at the base of the brain. These small granulations are conspicuously numerous in the choroid plexus, and cause great irritation in the neighboring parts. The irritation is followed by an exudation of greater or less extent into the ventricles. Accompanying this there is also a fibrinopurulent exudation between the pia mater and the cerebral convolutions at the base of the brain, notably in the fissures of Sylvius, but at times covering the whole convexity. This inflammatory exudation is usually confined to the meninges, but it may extend into the tissue of both brain and cord and over the upper surface of the cerebellum. A marked characteristic of the disease, and one which tends to distinguish it from other forms of meningitis, is the extension of the exudation from the meninges around the sheaths of the cranial nerves. The nerves apt to be affected are the auditory, optic, and trifacial. The amount of exudation is not proportionate to the number of tubercles. The ventricles are sometimes so distended as to burst the septum. Pressure is thus brought upon the central portions of the brain, involving especially the optic thalamus, the corpus striatum, and the corpus callosum.

In a certain proportion of the cases the miliary tubercles are more or less generally distributed over the surface of all parts of the brain, and the individual tubercles are of approximately the same size. In these cases there is much less fibrinous or purulent exudate along the course of the vessels. The lesions in this type probably represent a miliary tuberculosis of the meninges rather than a tuberculous meningitis, and are part of a general acute miliary tuberculosis. In other cases there is usually a part of the brain where the tubercles are more numerous and of larger size than in other parts. Sometimes in this region there are actual areas of caseation. This region represents the original focus of meningeal infection from which the bacilli have gradually spread through other parts of the pia mater. The farther one gets from this area the less numerous are the tubercles and the smaller are their size, until finally areas are reached in which no tubercles are found. In this type, fibrinous and purulent exudate is always very marked, particularly in the original area of infection and in the adjacent regiont. This type represents an original focal infection of the meninges, from which the disease is spread.

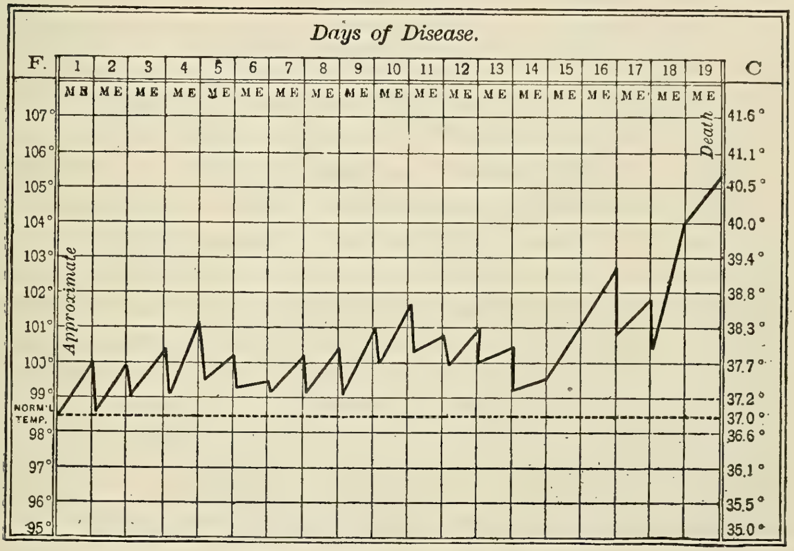
SYMPTOMS.—In general the characteristics of tuberculous meningitis are a gradual onset, a gradually increasing drowsiness leading eventually to stupor, and an absent or moderate febrile reaction.

In the great majority of cases the onset is gradual. In many cases in the first two years of life, however, the history obtained from the parents is that of an abrupt onset. It is probable that in these cases the early manifestations of the disease, which are not so noticeable in infants as in older children, were overlooked. Many cases are apparently in perfect health up to the time when the first symptoms of cerebral irritation are seen. In some cases, however, there is a previous history of malnutrition, loss of weight, anemia, and digestive disturbances. The earliest nervous symptoms are a disinclination to play, which is often combined with constant fretfulness or irritability. At times there is a complete change in the disposition of the child, who becomes peevish, capricious, and in some cases, easily frightened. There is often a tendency to sleep more than is normal, although the child when awake seems as bright as usual. Sleep is apt to be restless and disturbed. Older children in this early stage may complain of dizziness, headache, or slight evanescent pain in the head. In some cases a slight staggering in the gait is seen; in some cases the sharp so-called hydrocephalic cry is heard at night. Usually during this stage of the disease vomiting is seen, but at times this does not occur until after the increasing drowsiness has begun. Vomiting is a particularly prominent symptom in infancy, and occurs without any apparent reference to the diet or to indigestion. The bowels are usually constipated. The temperature may be slightly elevated, but in many cases is normal.

The duration of these indefinite symptoms is very variable. In some abrupt cases only a few days may elapse before increasing drowsiness appears. In other cases these indefinite symptoms of cerebral irritation may last for two or three weeks without at any time being sufficiently severe to attract the attention of the parents.

The most characteristic symptom of tuberculous meningitis is an increasing apathy and drowsiness. The child gradually takes less and less interest in its surroundings and wants to sleep most of the time. It is aroused with greater and greater difficulty, and the drowsiness deepens into stupor. The vomiting is very apt to remit after the stage of drowsiness and stupor has become established. The appearance of the child is now characteristic. Its eyes are open and staring, but it takes no notice of its surroundings and cannot be aroused. The pupils may be dilated or contracted. They react very sluggishly or not at all to light, and may be unequal. At times there is strabismus, ptosis, or facial paralysis. There may be hemiplegia, or a spastic paralysis of one or more limbs. A peculiar characteristic of the spasm or paralysis in tuberculous meningitis is that it is not constant, coming and going, or changing in distribution from day to day. There may be automatic movements of any of the extremities, or muscular twitchings may be noted. The fontanel is tense and bulging. There may or may not be rigidity of the neck, but this sign is a little more frequently absent than present in tuberculous meningitis. Opisthotonos is seen in some cases which have rigidity of the neck. The abdomen is often markedly retracted, giving the typical boat-shape; this appearance is most commonly seen in older children. The tâche cérébrale can usually be obtained by drawing the finger nail along the skin of the abdomen. The reflexes are very variable; the knee-jerks are usually increased in the early stage, and absent after the condition of stupor has become marked, but a change in the reflexes from day to day is quite characteristic of the disease. As stupor develops, the pulse which may previously have been rapid, usually becomes slow, irregular, and often intermittent. The respiration almost always shows marked irregularity and often approaches the Cheyne-Stoke's type. Ophthalmoscopic examination usually reveals the presence of choked disc, and often tubercles may be seen in the choroid.

CHART 26



Tubercular meningitis. Male, 4 years old.

The duration of this stage of the disease is variable, usually being from three to ten days. When the final stage is reached there is usually more or less complete musclular relaxation, the child loses the power of swallowing, and the pupils become widely dilated and do not react to light.

The temperature now rises rapidly to 104° F. and sometimes even to 106° F. or 107° F. The pulse becomes very rapid and feeble, often reaching 160 to 180 per minute. The respiration while still irregular, becomes more and more rapid and shallow. Even the duration of this final stage is very variable. Sometimes death occurs very quickly, while in other cases the patient may live for several days in a condition of such extreme exhaustion that death is hourly expected.

Death may occur from exhaustion in complete coma, or sometimes continuous convulsions lasting for many hours may precede the fatal ending. The rapid rise in the temperature generally means that death is not far off.

The above description applies to the most typical cases of tuberculous meningitis. The clinical manifestations of the disease, however, are so extremely variable that it is difficult to give an adequate description which shall cover all the various symptoms and types which are seen. In some cases, not very common, the first symptom noted is a rather prolonged convulsion, or an attack of several short convulsions. This may be followed by a very rapid development of drowsiness and stupor without there being any vomiting or other prodromal symptoms. In other cases, the development of the stage of drowsiness is preceded by a period of irritative symptoms which may last several days. These may be so marked as to suggest that the disease is epidemic cerebrospinal meningitis rather then the tuberculous form. There may be severe frontal headache, photophobia, attacks of screaming, hyperesthesia of the skin, and other symptoms of marked cerebral irritation. The neck may be rigid and tender, with retraction of the head. There may be rigidity of the extremities with the presence of Kernig's sign. The reflexes may be markedly exaggerated. The pupils may be normal or contracted, and there may be nystagmus. These irritative symptoms are apt in tuberculous meningitis to alternate with periods of marked apathy and dulness. The pulse is usually accelerated in this stage, but at times it is slow and weak. Careful observation will usually show an irregularity of the respiration, particularly during sleep. The temperature may be somewhat elevated in this irritative stage, but rarely goes above 100.5 unless there is a coexistent tuberculous bronchopneumonia. This irritative stage may last a number of days, but is eventually succeeded by the more typical stage of stupor.

It is only in comparatively rare cases that localizing symptoms of spasm or paralysis appear in the early stages of the disease. They are not usually seen until the stage of drowsiness and stupor is well advanced. Nevertheless, it should be remembered that the course of the disease is subject to the greatest variations, especially in older children. Quite often in children over two years old the steady advance of the disease is interrupted by periods of remission. In such cases, a child after having advanced into the stage of stupor, may recover consciousness and may even be able to sit up in bed and play with its toys. This is extremely deceptive, leading to false hope on the part of the parents that an error in diagnosis has been made. If, however, the disease is really tuberculous meningitis, such remissions do not last longer than two or three days, the child sinking again into stupor, and the fatal progress of the disease goes on. The entire duration of the disease is very variable. It is rarely more than three weeks, and in infants it is often much shorter than this.

DIAGNOSIS. Laboratory Diagnosis.—A positive diagnosis can only be made by means of lumbar puncture, but it is rare that the symptoms of the disease are sufficiently suggestive to warrant lumbar puncture until the stage of increasing drowsiness has appeared. The fluid obtained by lumbar puncture shows the following characteristics:

It is usually perfectly clear, although in very rare instances it may be slightly cloudy. It is almost invariably under increased pressure. Upon standing there is usually a fibrinous deposit, although this is sometimes absent. The cell count usually lies between 100 and 300 cells per cubic millimeter. A differential count of the cells shows that the great majority, usually over 95 per cent., are of the mononuclear variety. Exceptional cases are seen in which the polymorphonuclear cells are in excess.

Tubercle bacilli are almost invariably present in the cerebrospinal fluid, although their finding requires careful and often prolonged examination. The fluid taken for this examination should be preferably the last 15 or 20 c.c. which flow during the lumbar puncture. Great care should be taken not to shake the tube, but it should be allowed to stand, preferably in an incubator, for about twelve hours. At the end of this time, the fibrinous web which has formed in the fluid should be carefully removed entire with a platinum loop, and should be all spread out upon the cover-glass. If the bacilli are not found, another tube may be centrifugalized, and search may be made in cover-glass preparations from the sediment. The average physician is likely to be greatly deceived by the usual published accounts as to the per cent. of cases in which tubercle bacilli can be found in the cerebrospinal fluid of tuberculous meningitis. There are many reports which state that bacilli can be found in 80, 90, or even 95 per cent. of the cases. This is undoubtedly true if in each case the search is continued until the bacilli are found, no matter how long it takes. These reports do not make any mention of the length of time which was consumed in finding the bacilli in this large percentage of cases. It is all a question of how prolonged a search be made. The prolonged examinations on which these reports are based can be made in a research laboratory, or in any hospital with special laboratory workers. They cannot, however, be made by the ordinary practitioner, who may not be able to devote more than half an hour to the examination of a cover-glass. If no more time than this be devoted to the examinations, in my experience, no matter how careful the technic employed, the bacilli will not be found in more than 50 per cent. of cases of tuberculous meningitis.

Under such circumstances the question at once arises as to the significance of the increased cell count in the diagnosis of tuberculous meningitis. In the majority of cases, a diagnosis based on an increase of mononuclear cells in the cerebrospinal fluid will be accurate, but the physician must be familiar with the other conditions which may give an increased cell count in the cerebrospinal fluid. The commonest conditions in which an increase in the cell count is seen in childhood are *poliomyeloencephalitis*, and the condition of toxic meningeal irritation which is sometimes referred to for convenience under the name of *meningismus*. In poliomyeloencephalitis an increase in the cell count is constant in the acute stage of the disease. Furthermore, the increase may be quite as great as is seen in typical cases of tuberculous meningitis, so that there is no essential difference in the characteristics of the cerebrospinal fluid in the two diseases. Fortunately, however, it is only in rare cases that the differential diagnosis lies between these two conditions; poliomyeloencephalitis with its acute febrile onset, and the comparatively rapid development of a distinct and permanent paralysis does not often resemble typical cases of tuberculous meningitis. Nevertheless, in the preparalytic stage of poliomyeloencephalitis, meningeal symptoms may be present, and if in such cases the cerebrospinal fluid obtained at lumbar puncture shows an increase in the cell count, the physician may be in doubt as to the diagnosis. In such cases the question can only be settled either by the finding of the tubercle bacillus upon prolonged search of the cerebrospinal fluid, or by the final development of the paralysis characteristic of poliomyeloencephalitis.

In the majority of cases of meningismus the cell count is normal. Occasionally, however, an increased cell count is observed in connection with a severe toxic irritation of the meninges. This is due to the fact that the meninges react to toxic irritation in the same way as other serous membranes, and that a severe injury will cause some cellular exudate. The cell count in meningismus, however, is not increased in so marked a degree as in tuberculous meningitis. It is true that in some cases of tuberculous meningitis the cell count is only moderately increased. The highest cell count which I have seen in meningismus is 60 cells per cubic millimeter, while the lowest cell count which I have seen in tuberculous meningitis is 30 cells per cubic millimeter.

The following tables based on the writer's records will give an approximate idea of the average cell count in tuberculous meningitis:

TABLE 76

CELLS PER CUBIC MILLIMETER	NUMBER OF COUNTS
0- 10	0
10- 50	5
50-100	12
100-200	25
200-400	25
over 400	4

This table shows the amount of increase above the normal usually found. From another point of view, the following table shows the percentage of cases in which the increase reached certain figures:—

TABLE 77

CELLS PER CUBIC MILLIMETER	PERCENTAGE OF CASES
Over normal	100 per cent
Over 50	93 per cent
Over 100	76 per cent
Over 200	40 per cent
Over 300	15 per cent
Over 400	5 per cent

From these results the inference may be drawn that an increase in the number of cells per cubic millimeter found in the cerebrospinal fluid in tuberculous meningitis is constantly present and is usually considerable. There is, indeed, quite a wide margin, of about 20 cells per cubic millimeter, between the upper normal limit and the lower limit found in tuberculous meningitis. This is diagnostic in distinguishing tuberculous meningitis from patients who are not ill.

Other diseases in which the cell count in the cerebrospinal fluid is increased are too rare to enter into consideration, or else are diseases in which there could be no suspicion of tuberculous meningitis. Among them are the following: Syphilis with active secondary lesions, syphilitic meningitis, syphilis of the central nervous system, tabes dorsalis, general paresis, mumps, herpes zoster.

The following table, based on a series of sixty cases taken from the writer's records, will show the frequency of occurrence of the various characteristic signs and symptoms in tuberculous meningitis.

TABLE 78

SYMPTOMS		NUMBER OF CASES	SYMPTOMS		NUMBER OF CASES
Onset	Sudden	9	Convulsions	Early	8
	Medium	13		Developed	12
	Gradual	37		Absent	40
Temperature	High	11	Vomiting	Present	41
	Moderate	18		Absent	19
	Low	19	Pulse	Slow	15
Unconsciousness	Early	2		Rapid	43
	Medium	15	Cheyne-Stokes Breathing	Present	18
	Late	41		Absent	42

TABLE 78—*Continued*

SYMPTOMS	NUMBER OF CASES	
Headache	Present	17
	Absent	42
Rigid Neck	Early	7
	Developed	29
	Absent	23
Kernig's sign	Early	7
	Developed	21
	Absent	31
Knee-jerks	Exaggerated	15
	Normal	20
	Absent	24
Localizing Neurologic signs	Early	2
	Developed	19
	Absent	39
Babinski	Present	19
	Absent	35

SYMPTOMS	NUMBER OF CASES	
Leukocytosis	Present	10
	Absent	8
Tuberculin Reaction	Present	8
	Absent	5
Spinal Fluid	Clear	51
	Slightly cloudy	7
	Cloudy	0
	Bloody	2
Intradural Pressure	Increased	42
	Normal	16
Predominating Cells	Mononuclear	60
	Polynuclear	0
Fibrin	Present	42
	Absent	16
Bacilli	Found	15
	Not found	45

Total Duration

2- 4 days	3
5- 7 days	4
8-14 days	15
15-21 days	15
22-28 days	11
Over 28 days	8

The Ross-Jones, Nonne, and Noguchi tests for globulin are positive in the cerebrospinal fluid of cases of tuberculous meningitis. They are useful only in distinguishing the fluid from normal fluid, but are of no value in distinguishing between the various forms of meningitis.

The general laboratory methods used in the diagnosis of tuberculosis are of comparatively little value in tuberculous meningitis. There is a marked difference of opinion among various observers as to the constancy of a positive von Pirquet tuberculin test in this disease. Some authorities state that the variations in the reports of different observers are due to differences in technic, or in the quality of the tuberculin employed. I am convinced, however, that there is a very marked variation in the constancy of the immune reaction of tuberculosis. Nothing else can explain the discrepancy in the reports of various observers. Furthermore, even here in Boston, there are great variations in the percentage of cases which show a positive tuberculin reaction from year to year, even when the same technic and tuberculin are employed. A positive reaction is of great assistance in the diagnosis of tuberculous meningitis, when it can be obtained. A negative reaction, on the other hand, is of comparatively little value. It certainly does not exclude the possibility of tuberculous meningitis, particularly in poorly nourished or emaciated infants.

It is only in very rare instances that tubercle bacilli can be obtained from the sputum of infants with tuberculous meningitis. The presence, however, of a positive d'Espine's sign, or of a roentgenogram of the chest which shows a distinct enlargement of the peribronchial lymphnodes, is confirmatory evidence of the tuberculous nature of the disease.

CLINICAL DIAGNOSIS.—If the diagnosis of tuberculous meningitis can only be positively made from the results of lumbar puncture, the practical question which arises is, when in the course of the disease is lumbar puncture indicated? In the early stage of the disease, which is characterized only by the vaguest prodromal symptoms, it is impossible to suspect tuberculous meningitis, and lumbar puncture is only to be considered when some definite symptoms have developed. The most suggestive symptoms of tuberculous meningitis in the order of their frequency are the following: Increasing or persistent drowsiness, repeated vomiting without apparent cause, obstinate constipation, irregular respiration, irregular pulse, convulsions, rigidity of the neck, and slight fever without obvious cause. Whenever a combination of any of these symptoms has developed, diagnostic lumbar puncture is indicated. Furthermore, at any time when the symptom complex suggests the possibility of either tuberculous meningitis or some other disease which ordinarily has symptoms which may appear in tuberculous meningitis, diagnostic lumbar puncture is indicated in order that a differential diagnosis may be made. This is particularly true in cases characterized by the onset of irritative symptoms, which are strongly suggestive of some form of meningitis.

DIFFERENTIAL DIAGNOSIS.—There are various fairly common conditions which have to be considered in the differential diagnosis of tuberculous meningitis.

Gastro-Intestinal Intoxication.—This condition frequently gives a clinical picture of vomiting and symptoms of cerebral irritation. When these symptoms resist dietetic treatment, the question often arises whether or not a beginning tuberculous meningitis is present. Lumbar puncture is indicated in such cases.

Acidosis.—This condition frequently resembles the more acute cases of tuberculous meningitis, being characterized by an onset with obstinate vomiting followed by an increasing apathy, which may deepen into stupor. While most cases of acidosis develop much more rapidly than tuberculous meningitis, yet a positive differential diagnosis cannot be made from the clinical symptoms. The presence in the urine of a marked reaction for acetone and diacetic acid is strongly suggestive of the diagnosis of acidosis, but sometimes the acetone bodies are found in the urine in cases of tuberculous meningitis characterized by prolonged vomiting. The development of any localizing cerebral symptoms is suggestive of tuberculous meningitis. The respiration in acidosis is usually regular, although quickened and deepened. In many cases lumbar puncture is required to differentiate between the two conditions.

Other Forms of Acute Meningitis.—Typical cases of tuberculous meningitis are quite different in their symptoms and course from typical cases of epidemic cerebrospinal meningitis, or of acute meningitis due to other organisms. In general the presence of an acute onset, high fever, rigidity of the neck, Kernig's sign, marked signs of cerebral irritation, and an early development of apathy or stupor, points toward one of the other forms. Nevertheless, atypical cases of tuberculous meningitis are frequently seen which in their symptoms closely resemble the epidemic form. This is particularly true in infancy. A differential diagnosis can only be made by lumbar puncture, which is always indicated in cases which are characterized by the rapid development of distinct meningeal symptoms.

Poliomyeloencephalitis.—This is the only condition in which the differential diagnosis cannot be made by lumbar puncture, unless tubercle bacilli are found in the cerebrospinal fluid. It is quite easy to distinguish all types of poliomyeloencephalitis from typical cases of tuberculous meningitis, on account of the acute febrile onset which characterizes the former disease. Gradually increasing drowsiness is not often seen in poliomyeloencephalitis. Either there are no symptoms in the prodromal stage suggesting a disease of the central nervous system, or else the symptoms are those of meningeal irritation, which suggest epidemic meningitis rather than the tuberculous form. These cases with meningeal symptoms, however, closely resemble the more acute atypical cases of tuberculous meningitis. If the cerebrospinal fluid simply shows an increase in the cell count without the presence of tubercle bacilli, the diagnosis may be in doubt until either the bacilli are found or until a characteristic paralysis develops.

Poliomyeloencephalitis is characterized by the development of a definite and constant paralysis which in the majority of cases is of the flaccid spinal type, but which may be of the spastic cerebral type in the encephalitic form of the disease. The important distinguishing point is that in poliomyeloencephalitis the paralysis develops fairly early in the course of the disease, and once having developed, is constant in its distribution. When paralysis develops in tuberculous meningitis, it is a comparatively late symptom, is almost invariably of the cerebral spastic type and shows a marked tendency to change its severity and distribution from day to day.

Typhoid Fever—This disease is often confounded with tuberculous meningitis, on account of its gradual onset and the absence of definite signs on physical examination. The steadily increasing febrile reaction of typhoid fever is usually very different from the moderate or absent fever seen in the early stages of typical tuberculous meningitis. Some cases of typhoid fever in early life are characterized by symptoms of meningeal irritation, and these make the differential diagnosis all the more difficult on clinical grounds. The diagnosis of typhoid fever eventually depends upon the appearance of enlargement of the spleen, rose spots, or a positive widal reaction, while the diagnosis of tuberculous meningitis depends upon the absence of the typhoid temperature curve and upon the eventual development of increasing signs of cerebral disease. The count of leukocytes in the blood is not of very great value; while a diminished white count points toward typhoid, a normal white count may be seen in both diseases. I have seen cases in which a differential diagnosis was in doubt for a considerable time, although the most important differential point in the early stages is the character of the temperature curve. A lumbar puncture will at once enable the physician to distinguish between the two conditions.

Solitary Tubercle of the Brain.—This condition is usually of much more gradual development than tuberculous meningitis. Vomiting occurs at longer intervals. Convulsions occur in both conditions, but in solitary tubercle they occur at intervals for a long time before there is any increasing apathy or drowsiness. In tuberculous meningitis convulsions are usually comparatively late manifestations. In most cases with solitary tubercle the signs of cerebral irritation are apt to suggest a localized lesion, while in tuberculous meningitis they suggest a general widespread disease. The principal differentiating point, however, is the much more gradual development and chronic course of solitary tubercle. Nevertheless, I have seen cases which on account of the acuteness of their course were diagnosed clinically as tuberculous meningitis, but which showed only solitary tubercle at autopsy. Lumbar puncture is always indicated when there is a doubt between these two conditions. The cerebrospinal fluid in solitary tubercle is usually under increased pressure, but ordinarily shows neither fibrinous clot, increase in the cell count, nor tubercle bacilli.

Cerebral New Growth.—As the symptoms of this disease are practically the same as those of solitary tubercle, the differential diagnosis is made in the same way.

Syphilitic Meningitis.—This is a very rare condition in early life. Its diagnosis depends upon a combination of the symptoms of a chronic meningitis with the history and general symptoms of syphilis. The diagnosis should be confirmed by a positive Wassermann reaction.

Nephritis with Uremia.—This condition is rarely mistaken for tuberculous meningitis. It is very uncommon in infancy and early childhood. The diagnosis depends upon the result of the examination of the urine.

Malaria.—The manifestations of malaria in infancy and early childhood are so irregular that they may simulate those of almost any disease. I have seen cases of malaria in infancy mistaken for tuberculous meningitis. The differential diagnosis depends upon the results of the examination of the blood and of lumbar puncture.

PROGNOSIS.—The prognosis of tuberculous meningitis is bad, in fact almost absolutely unfavorable. There have been a few reported cases of recovery from tuberculous meningitis in which tubercle bacilli were found in the cerebrospinal fluid. Nevertheless, instances are so extremely rare that the physician is not justified in holding out more than the faintest hope to the parents. It is known that some cases of solitary tubercle of the brain may terminate in recovery, and while in this condition tubercle bacilli are not usually found in the cerebrospinal fluid, there is a possibility of their presence when the tubercle grows to the meningeal surface. I have seen one case in which tubercle bacilli were found in the fluid, but which showed no tuberculous meningitis at autopsy, but only solitary tubercle of the brain. It is possible that some of the very few recorded instances of recovery from tuberculous meningitis may have been cases of solitary tubercle. I usually tell parents that there is very slight hope for the child, and that there are only two possibilities, one being that my diagnosis is wrong, the other being based on the very rare instances recorded of recovery. Parents should, however, be made to understand that the chances against the child are fully one hundred to one.

TREATMENT.—It is obvious that there can be no specific treatment. The physician's efforts should be directed at keeping the child as comfortable as possible, and at meeting such symptoms as may arise. I do not believe that it is advisable to keep an ice-bag to the head, as this usually appears to increase the discomfort of the child, and of course has no influence upon the disease. The main problem is one of nutrition, and most cases have to be fed by gavage before the fatal ending is reached. Pain is rarely severe enough to require medication. The various symptoms of nervous irritation may be combatted by means of bromides, which, if the stomach is intolerant, may be given by rectum. When the fatal ending is approaching, stimulation is of no value, and it is better to leave the dying patient alone than to harass it with useless subcutaneous injections.

ACUTE ENCEPHALITIS

This is an acute inflammation of the cerebral tissues due to infection.

ETIOLOGY.—It is now known that the great majority of cases of acute encephalitis seen in infancy and early childhood represent simply a particular manifestation of the specific infectious disease poliomyeloencephalitis. Cases of this type have been described under the encephalitic type of infantile paralysis. (Volume I, p. 653.) There are, however, a certain number of cases which are apparently not caused by the virus of poliomyeloencephalitis. The separate nature of the infection in these cases is recognized by the fact that they usually occur in the course of some recognized infectious disease. Acute encephalitis is most frequently seen in association with scarlet fever, measles and mumps, but may occur as a complication of any of the infections. In some cases the encephalitis is apparently primary, but it is probable that in these cases it is really secondary to some primary infection which has been overlooked. The disease is most common in infancy, and only in rare instances are cases seen in later childhood. Little is known of the microörganisms which produce these forms of acute encephalitis, because the lesions are usually localized and not fatal.

PATHOLOGICAL ANATOMY.—As the disease is usually not fatal, little is known of the pathological anatomy in the acute stage. Most of the fatal cases which have given the clinical picture of acute encephalitis, have proved at autopsy to be poliomyeloencephalitis. What we know of the lesions of the brain substance in most cases depends upon the autopsy findings in long standing cases of cerebral paralysis. The pathological changes in such cases are mainly atrophy and sclerosis, followed by secondary degeneration in the spinal cord, but these lesions represent only terminal conditions. The acute inflammation in secondary encephalitis is probably very similar to that of poliomyeloencephalitis, the only difference being etiological.

SYMPTOMS.—The symptoms closely resemble those of the encephalitic type of poliomyeloencephalitis. There is usually an acute onset with high fever, vomiting, often convulsions, followed by delirium or stupor. These general symptoms of acute infection continue for a variable time, usually from one to three days, before paralysis is seen. The temperature usually lies between 101°F and 103° F. Loss of consciousness may last for several days, and paralysis may not be discovered until the patient returns to consciousness.

In rare cases the loss of consciousness deepens into profound coma, with disturbance of respiration and death. In the majority of cases there is either no loss of consciousness, or consciousness is quickly regained.

The paralysis usually takes the form of a typical cerebral hemiplegia. It usually involves both the arm and the leg, and sometimes the face. Disturbances of sensation are rarely present, and if so are usually transient.

The paralysis of the arm and leg is apt to be complete at first, but improvement is usually rapid for the first few weeks after the acute stage. The paralysis is of the spastic type with increased reflexes. The convulsions at the onset may be repeated for the first few days, but each convulsion is rarely so long in duration as in hemorrhage; they may be either general or unilateral. A slight ankle clonus may be present on the affected side. When the lesion is on the left side of the brain, motor aphasia is sometimes seen, but this is not common.

The subsequent course of these cases is very similar to that seen in acquired cerebral hemorrhage. Improvement usually begins first in the leg and later in the arm, and is often quite rapid. After a few weeks the patient usually regains the power of walking, although there is a variable amount of residual paralysis, which causes a dragging of the affected leg. The final outcome is very variable. Sometimes the leg recovers completely in other cases a variable amount of spastic paralysis persists, which causes limping or a halt in the gait. Recovery in the arm is apt to be less complete, and in the course of one or two years contractures are very likely to develop. Contractures may also develop in the leg in severe cases. In old cases there is more or less atrophy of the paralyzed limbs combined with a variable amount of rigidity.

In the majority of cases this type of acute encephalitis leaves behind no permanent impairment of the mental condition. Occasionally, however, when the disease occurs in early infancy a certain amount of mental deficiency follows, which makes such cases rather difficult to distinguish from cases of congenital developmental lesions of the brain. The most common sequel of secondary encephalitis is a tendency toward occasional convulsions, which may persist not only throughout childhood, but even continue into adult life. The convulsions may be general, and in such cases the condition is only to be distinguished from epilepsy by the presence of a residual cerebral paralysis. In other cases the epilepsy is of the Jacksonian type, the convulsions being confined to the paralyzed limb or limbs without complete loss of consciousness. In some cases posthemiplegic movements of a choreic or athetoid nature are seen.

DIAGNOSIS.—In the acute febrile stage, before the appearance of paralysis, the diagnosis cannot be made. When symptoms of cerebral disturbance are sufficiently pronounced to warrant lumbar puncture, the examination of the cerebrospinal fluid will give evidence as to whether the disease is poliomyeloencephalitis, or something else. The former disease is recognized by the characteristic increase in the cell count, which is not present in secondary encephalitis. A lumbar puncture is also useful in excluding the various forms of meningitis.

After the cerebral paralysis has appeared the physician must consider three acute conditions which lead to hemiplegia. These are in the order of frequency,—poliomyeloencephalitis, acute secondary encephalitis, and hemorrhage. Meningitis rarely leads to so definite and constant a paralysis as is seen in the three conditions just enumerated.

Poliomyeloencephalitis.—The cases with an acute febrile onset followed by the rapid development of a definite and constant paralysis, should be diagnosed as the encephalitic type of poliomyeloencephalitis rather than as acute secondary encephalitis, unless the cases occur in the course of a recognized acute infection. The diagnosis of infantile paralysis is confirmed by lumbar puncture. If the cerebrospinal fluid does not show an increase in the cell count, acute secondary encephalitis may be suspected.

Cerebral hemorrhage.—Both cerebral hemorrhage and acute encephalitis are usually described together under the term *cerebral paralysis*, because these are the two commonest lesions which give the clinical picture of spastic paralysis of a hemiplegic type. The principal point in the differentiation between the two conditions is that acute encephalitis is characterized by a marked febrile onset which in every way suggests that of an acute infection, whereas cerebral hemorrhage usually develops without fever. If, however, the patient is already suffering from acute infection, the fever may be due to the primary disease, and it is difficult to say whether the cerebral lesion is encephalitis or hemorrhage. It should be remembered that encephalitis is more common than acquired hemorrhage. The majority of cases of cerebral paralysis in early life due to hemorrhage are of congenital origin. Cases of acquired hemorrhage may sometimes be recognized by the presence of blood in the fluid obtained by lumbar puncture. When the patient is first seen after convalescence from the acute stage, and presents only the residual cerebral paralysis, the etiological diagnosis depends upon how accurate a history of the acute attack can be obtained, but is not essential for purposes of treatment.

PROGNOSIS.—Acute secondary encephalitis is rarely fatal. As far as the residual paralysis is concerned, the prognosis is very favorable. In individual cases there is usually marked improvement. The extent of the improvement finally attained depends more upon how rapidly it begins and progresses than upon the extent and distribution of the paralysis. Facial paralysis usually disappears entirely. In hemiplegia, entire recovery may occur, but usually there is at least a slight limp or dragging of a leg. In other cases, more or less permanent paralysis remains in the arm, leg, or both, with eventual contraction and deformity. The liability to the subsequent development of Jacksonian epilepsy or generalized convulsions must always be remembered, and this sequela is, if anything, more likely to occur in mild cases which show almost complete recovery than in more severe ones.

TREATMENT.—During the acute stage the treatment is that of any acute disease of similar severity with signs of irritation of the cortex of the brain. The child should be kept absolutely quiet in bed, with an ice-bag or Leiter's coil applied to the head. If there are convulsions, they should be treated as described under that heading. If signs of cerebral irritation are severe, the bromides should be used freely. During the stage of improvement, after the acute symptoms have subsided, the treatment is just the same as that described for the convalescence of poliomyeloencephalitis. The child should at first be kept absolutely quiet, and then should gradually be allowed to resume whatever activity is possible. Massage is useful for the residual paralysis. Electricity has not given proof of value sufficient to warrant its use. The physician should make sure that the paralyzed limbs are kept in a normal position and should use every effort to prevent the occurrence of early contracture and deformity. After the limit of improvement has been reached, the treatment is orthopedic, with massage, training, apparatus, and sometimes tenotomy.

CEREBRAL ABSCESS

Cerebral abscess is a localized purulent encephalitis and is probably always secondary to suppurative disease elsewhere. It may arise from a suppurative condition of the scalp, but its most common source is some purulent disease of the ear or other adjacent structures. Its usual location is in the temporal lobe, just above the tegmen tympani, or in the cerebellum. It is also found as a sequel to traumatism of various kinds resulting in suppuration and in general pyemia, and it may follow direct traumatic injury to the brain. Cerebral abscess is usually single, except when it is produced by pyemia. The abscess may occur in any part of the brain. Cerebellar abscess is not uncommon.

SYMPTOMS.—A cerebral abscess may exist for a considerable time without producing any symptom which can be recognized during life. In cases in which suppurative disease of the ear exists, a cerebral abscess may be suspected when, in addition to the temperature, which would naturally be raised from this process, the child's general condition becomes worse without any apparent cause, and when indefinite symptoms, such as mental dulness and irritability, arise. The temperature and marked leukocytosis may also suggest the presence of imprisoned pus, while the other symptoms suggest the probability of cerebral disease, in cases in which the pus cannot be found elsewhere. Cerebral abscess, however, may rarely exist without rise of temperature. It is apt to be slow in its progress and to cause general constitutional rather than local symptoms. Local symptoms produced by the presence of cerebral abscess, as a rule are absent, but may be manifested by sensory aphasia if the left temporal lobe is involved, or, in the case of cerebellar disease, by hemiataxia, nystagmus, a tendency to walk or fall to one side, diminished or increased tonicity of the muscles on the same side, the symptoms varying according to whether the lesion acts in an irritative or paralytic manner. General symptoms of brain abscess are represented by headache, vertigo, dimness of vision caused by optic neuritis, mental dulness, vomiting, slow pulse, foul breath, and convulsions followed later by coma. When the abscess bursts into the ventricles, symptoms of collapse appear, and death rapidly follows. Tremor and convulsions may occur in cases of cerebral abscess, but neither of them should be considered as in any way diagnostic of this condition. An abscess of the brain may remain latent for a long time and suddenly become active.

PROGNOSIS.—The prognosis is very unfavorable unless the disease can be reached surgically.

TREATMENT.—The treatment should be operative if the diagnosis can be made with certainty.

SEPTIC THROMBOSIS OF THE CEREBRAL SINUSES

This condition may exist as a part of an acute meningitis, and in such cases presents no characteristic symptoms. The most common cause apart from meningitis is otitis. The thrombosis of the sinus occurs usually as a complication of old and neglected cases of otitis. It should be remembered, however, that septic sinus thrombosis is much less frequently associated with disease of the ear in children than in adults. The lesion may also be secondary to traumatism, or to any septic process involving any of the tissues adjacent to the brain, such as the scalp, nose, orbit or pharynx.

PATHOLOGICAL ANATOMY.—In the cases which complicate otitis there is usually, but not always, necrosis of the petrous portion of the temporal bone. In these cases it is the lateral sinus which is the site of the septic thrombosis. In other conditions the seat of the thrombosis depends upon that of the original disease. With a septic process involving the cranial bones or scalp, the longitudinal sinus is affected. If the primary disease is at the base of the skull, in the orbit, in the pharynx, in the jaw, or in the nose, the cavernous sinus will be affected. In any case, the sinus becomes the seat of a clot which may undergo puriform softening with a general pyemia and the development of secondary pyemic abscesses in various parts of the body. In other cases, the suppuration of the clot leads eventually to an acute meningitis.

SYMPTOMS.—The general symptoms closely resemble those of acute meningitis. There is fever, headache, and sometimes vomiting. On the other hand, rigidity of the neck, Kernig's sign, and other signs of meningeal irritation are often absent. The most typical symptoms of septic sinus thrombosis are chills, which recur at varying intervals. The temperature is usually very high and shows very wide fluctuations on the four-hourly chart. If the disease takes the pyemic type, the various symptoms of metastatic abscesses in various parts of the body are seen.

The local symptoms vary according to the sinus affected, but are generally rather indefinite. When the lateral sinus is involved, the process may extend to the jugular vein, which may be felt in the neck as a hard cord. When the longitudinal sinus is involved, there may be dilatation of the temporal and frontal veins; sometimes there is cyanosis of the face, or epistaxis. When the cavernous sinus is affected, the most characteristic symptom is protrusion of the eyeball on the affected side with edema of the eyelid. In such cases opthalmoscopic examination shows that the retinal veins are enlarged and tortuous, and they may contain thrombi.

The course of the disease is very variable. The duration may be from a few days to several weeks.

DIAGNOSIS.—The most important point in the diagnosis is the recognition of dilatation of the superficial veins which empty into the sinus affected. The palpation of a thrombosed jugular vein is the most important point in the diagnosis of thrombosis of the lateral sinus. When these localizing signs are absent, the physician is confronted with a picture which in general resembles an acute meningitis. Lumbar puncture will exclude meningitis if none be present. The recognition of the fact that the symptoms are produced by septic sinus thrombosis then depends upon the finding of the primary seat of inflammation. When the disease takes the pyemic type with recurrent chills and metastatic abscesses, the diagnosis becomes more clear.

PROGNOSIS AND TREATMENT.—The prognosis is very grave. In the majority of cases death takes place from acute meningitis, pyemia, or cerebral abscess. The only hope is in surgical treatment. The lateral sinus is most accessible to surgical operation. The operation on the superior longitudinal sinus is very much more difficult. The cavernous sinus is practically inaccessible. Operation should always be urged when the diagnosis of septic thrombosis of the lateral sinus is clear.

MYELITIS

The commonest form of myelitis seen in early life is a lesion characteristic of the specific infectious disease poliomyeloencephalitis This disease has been described in Volume I. Second in frequency is the myelitis due to compression, which is usually seen in tuberculous caries of the spine. This has been described under the mechanical injuries of the central nervous system. There is, however, a form of myelitis which may occur as a complication of any of the acute infectious diseases. The process is in every way similar to acute secondary encephalitis, except that the infection involves the spinal cord instead of the tissues of the brain.

ETIOLOGY.—Secondary myelitis occurs most often as a complication or sequela of typhoid fever, scarlet fever, or diphtheria. It may occasionally follow any other of the acute infections. A certain number of cases have been attributed to exposure to cold, but in them the myelitis is probably also of infectious origin and secondary to a septic focus in some other part of the body. The disease is seen most often in the later years of childhood, but is nevertheless rare.

SYMPTOMS.—The onset may be gradual without any general constitutional disturbance suggesting an acute infection. In these cases only local symptoms are seen. In some cases the onset is acute with fever, pain, prostration, and the general constitutional disturbance characteristic of an acute infection. The characteristic symptoms of transverse myelitis are, spastic paralysis and anesthesia in all parts of the body supplied from the spinal cord below the level of the lesion, and pain or hyperesthesia in the parts of the body supplied from the spinal cord at the level of the lesion.

When transverse myelitis is situated in the cervical region, there is paralysis and anesthesia of the arms, legs and trunk. There is a general spasticity with a marked increase in all the reflexes. There may, however, be flaccidity and atrophy of some of the muscles of the arms. There is often pain referred to the arms, or areas of hyperesthesia in the arms. There is retention of urine followed by incontinence from overflow. When the lesion is high up in the cervical region, there is often contraction of the pupils, and in some cases optic neuritis is seen. Transverse myelitis of the cervical region is very dangerous to life, on account of the possibility of the paralysis affecting the muscles of respiration.

When the lesion is situated in the dorsal region, the symptoms are the same as those described above, except that the arms escape and the eye symptoms are lacking. There is often a zone of hyperesthesia about the body, and the so-called girdle pains.

When the seat of the disease is the lumbar region, the most prominent symptom is incontinence of urine and feces. There is anesthesia of the legs, but the paralysis usually takes a flaccid type with loss of knee-jerks, reaction of degeneration and muscular atrophy. Bed sores are very frequent in all cases of transverse myelitis.

In diffuse myelitis the local symptoms are a combination of flaccid and spastic paralysis, and of hyperesthesia and anesthesia. When a large part of the cord is involved there are usually marked trophic disturbances, bed sores, and so forth. The course of the disease is usually slow, and usually progresses steadily from bad to worse. Death is usually due to exhaustion, cystitis, septic infection from the bed sores, or to some intercurrent disease.

DIAGNOSIS.—The diagnosis depends upon the definite localization of the spinal paralysis. Poliomyeloencephalitis usually involves only the motor portions of the cord. It is sometimes difficult to distinguish a transverse myelitis from a tumor of the spinal cord, or from a hemorrhage into the cord.

PROGNOSIS.—The prognosis is very unfavorable. There is sometimes a partial recovery of function, but the majority of cases eventually die.

TREATMENT.—In the acute stage the treatment consists in the application of ice to the spine and in counter-irritation. Improvement has been reported following the use of iodid of potassium in large doses. The most important features of treatment are the nursing and cleanliness.

SOLITARY TUBERCLE OF THE BRAIN

This is a not uncommon secondary lesion of tuberculosis in early life. Many cases in infancy which come to autopsy as a result of tuberculous meningitis or acute general tuberculosis, have one or more solitary tubercles in the cerebral tissues. Occasionally, solitary tubercle is the only secondary manifestation of tuberculosis. The etiology and pathogenesis of this lesion have been discussed in the general article on tuberculosis in Volume I.

PATHOLOGICAL ANATOMY.—The lesion consists in a conglomerate tubercle which usually forms a caseous mass of varying size. These tuberculous masses may vary in size from that of a pea to that of a walnut, or even larger. At times there is only one tubercle, in other cases there are two or three. The tubercles may be situated in any part of the brain.

SYMPTOMS.—The symptoms of solitary tubercle usually resemble those described under intracranial tumors. There is nothing in the symptomatology which can enable the physician to distinguish a solitary tubercle from glioma or sarcoma. The reader is referred to the symptoms of new growths of the brain for a description of the symptoms usually seen in cases of solitary tubercle.

Occasionally more acute cases are seen in which the symptoms closely resemble those of a tuberculous meningitis.

DIAGNOSIS.—Solitary tubercle must be distinguished from cerebral new growths on the one hand, and from tuberculous meningitis on the other.

Cerebral New Growths.—It must be remembered that solitary tubercle in early life is a much more common cause of the symptoms of brain tumor than is a true new growth. The only way in which a differential diagnosis between the two conditions can be made, is through the establishment of the general diagnosis of tuberculous infection. As solitary tubercle of the brain is always secondary to tuberculosis in some other part of the body, its diagnosis depends largely upon the evidences of tuberculous infection which can be obtained. The diagnosis of tuberculosis in early life is often, however, difficult when, in addition to the brain lesions the only other lesions are those of the primary stage of the disease. The factors which enter into the general diagnosis of tuberculosis have been thoroughly discussed in the section on tuberculosis. In general, if d'Espine's sign and interscapular dulness are present, if a roentgenogram shows enlargement of the bronchial lymphnodes, if the von Pirquet reaction is positive, or if there are definite evidences of other secondary tuberculous manifestations, such as tuberculous bronchopneumonia, peritonitis, or cervical lymphadenitis, a diagnosis of tuberculosis can be made with a fair degree of certainty. When a diagnosis of infection with tuberculosis can be made with any of the methods used in the diagnosis of that disease, it is safe to assume that the symptoms of cerebral tumor are caused by a solitary tubercle.

Tuberculous Meningitis.—In cases in which the history is suggestive of tuberculous meningitis, the diagnosis depends upon the results of lumbar puncture. In solitary tubercle the cerebrospinal fluid is usually under increased pressure, but does not show any of the other characteristics of the fluid of tuberculous meningitis. The diagnosis of solitary tubercle is frequently made and confirmed at autopsy upon this basis. In any case giving the general evidences of tuberculous infection and with symptoms suggestive of tuberculous meningitis, but in which the results of lumbar puncture do not confirm the diagnosis of that disease, the probabilities are strongly in favor of solitary tubercle.

PROGNOSIS.—The prognosis is bad. Most cases eventually develop a fatal tuberculous meningitis. In some cases death occurs as a result of the gradually increasing injury to the cerebral tissues.

TREATMENT.—The treatment is purely symptomatic.

CEREBRAL SYPHILIS

Intracranial syphilis may be either congenital or acquired. The intracranial lesions are essentially the same in both forms. It must be remembered that the cerebral manifestations of syphilis are very rare in childhood as compared with adult life.

PATHOLOGICAL ANATOMY.—Intracranial syphilis may be divided pathologically into three forms: (1) Diffuse inflammation of the meninges or their neighboring tissues, (2) localized growths or tumors (gummata), and (3) syphilitic endarteritis. In the latter case there may be local dilatation or local occlusion of the blood vessels. These conditions are apt to occur simultaneously. When the dilatation reaches an advanced stage a thinning of the arterial walls results, which may lead to rupture of the blood vessels and hemorrhage. More common than hemorrhage, however, is occlusion of the blood vessels, which cuts off the blood supply and acts in the same way as thrombosis of the arteries due to other causes, producing more or less softening and disintegration of the cerebral tissues. The arteries of the base of the brain are the ones that are most frequently affected, and the secondary lesions involve parts of the brain supplied by them.

SYMPTOMS.—The symptoms dependent on these lesions vary in accordance with the pathological condition.

In syphilitic meningitis the principal symptoms are severe headache in various parts of the head, more or less constant, lasting for many days or even weeks, and frequently accompanied after a time by paralysis of some of the cranial nerves, especially of the third or of the seventh. As in other cases of meningitis, the optic nerves may also be affected, and the child may show the general symptoms of a severe intracranial affection, such as vomiting, dulness, headache, and localyzed paralyses.

The localized tumors or gummata present essentially the same symptoms as other forms of tumors of the brain.

The symptoms produced by syphilitic endarteritis vary according to the areas of the brain affected, but the most common ones are the various forms of paralysis of the extremities and various sensory disturbances.

DIAGNOSIS.—In regard to the diagnosis of cerebral syphilis in children, the symptoms differ greatly in different cases. The most characteristic group of symptoms, and one which is exceedingly suggestive of intracranial syphilis, includes attacks of organic paralysis, central in origin, occurring at intervals of days or months without known cause, and without marked symptoms of either tumor or tuberculosis.

The clinical diagnosis of syphilitic meningitis may be made from the occurrence of severe headaches, followed by paralysis of one or more of the motor cranial nerves, and occurring without marked rise of temperature.

Gummata present no especial symptoms differing from those of intracranial tumors. The presence of syphilitic lesions elsewhere or a history of syphilis in the parents is our principal ground for making the diagnosis.

Syphilitic endarteritis may be suspected when an acute affection in the neighborhood of the pons or medulla not produced by traumatism occurs in a syphilitic subject, or when acute symptoms suggestive of hemorrhage or embolism are present, and when no other probable cause can be shown.

The symptoms can never give positive proof of the existence of cerebral syphilis. The diagnosis depends upon other evidences of syphilis, obtained from the history or physical examination, and upon the results of the Wassermann reaction.

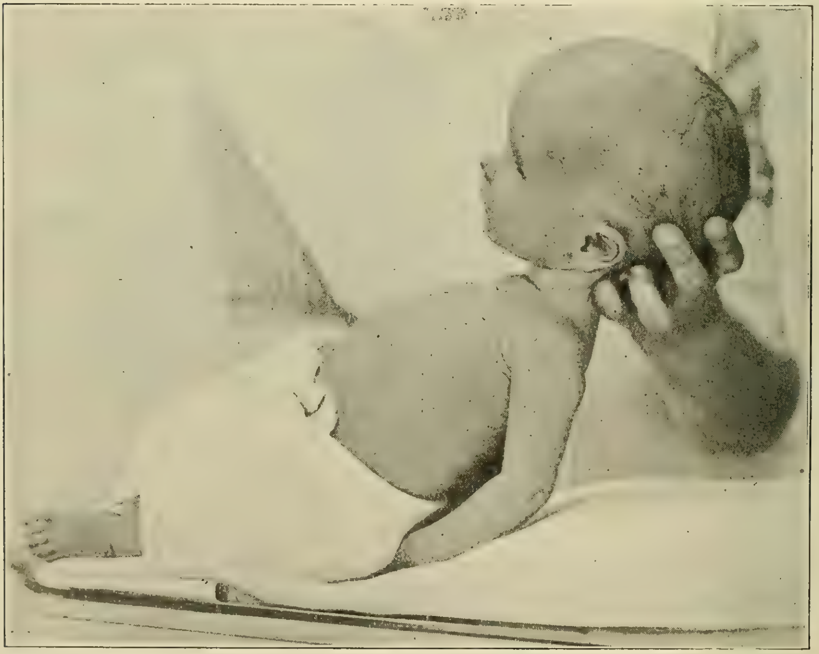
PROGNOSIS AND TREATMENT.—The results of antisyphilitic treatment in these cases are sometimes striking. The treatment should be pushed rapidly to the limit of tolerance.

VI. ORGANIC DISEASES OF UNKNOWN OR MISCELLANEOUS ETIOLOGY

HYDROCEPHALUS

By hydrocephalus is meant an accumulation of serous fluid in the cranium. This may occur either in the subarachnoid space (external hydrocephalus) or in the ventricles (internal hydrocephalus). When the accumulation of fluid represents the only pathological condition present, the term primary hydrocephalus is used, while when it follows in the course of other diseases it is termed secondary. Hydrocephalus may also be acute or chronic, congenital or acquired.

FIG. 281



Acute Hydrocephalus

ACUTE HYDROCEPHALUS. ETIOLOGY.—Acute hydrocephalus may be either external or internal, and is usually a combination of both. It is generally sudden in its onset, and while in some cases it may be idiopathic (meningitis serosa), in most cases it is secondary to a number of conditions, such as cardiac disease, pertussis, rachitis, neoplasms, tumors in the posterior fossa, acute inflammatory processes in the brain and its meninges, and acute febrile diseases, such as typhoid and pneumonia.

SYMPTOMS.—The amount of fluid in these acute cases is, as a rule, not large, 90 to 120 c.c. (3 or 4 ounces), and the symptoms are those which arise in meningitis when there is increased intracranial pressure, such as headache, vomiting, unequal pupils with sluggish reaction, double optic neuritis, rarely convulsions, and cerebral paralysis. In mild cases recovery may take place in a few weeks, but in the more severe forms the increased intracranial pressure may prove fatal.

Both the congenital and the acquired varieties of external hydrocephalus are so rare that they need not be more than mentioned as occurring in certain congenital malformations, in atrophy of the brain (hydrocephalus in vacuo), and following cerebral hemorrhage.

The general shape and circumference of the head in infancy and childhood vary normally in the individual to a considerable degree. On the other hand, when these variations in size pass a certain limit or are combined with certain nervous phenomena, they have a distinct pathological significance.

CHRONIC INTERNAL HYDROCEPHALUS. ETIOLOGY.—The chronic internal variety of hydrocephalus consists of a transudation into the cerebral ventricles, which causes a marked dilatation of those cavities. It may be *congenital* (intrauterine) or *acquired* (extrauterine) It may follow an operation for spina bifida or be produced mechanically by internal pressure from any cause. It may also represent the termination of an acute inflammatory process. In many cases the cause is wholly unknown.

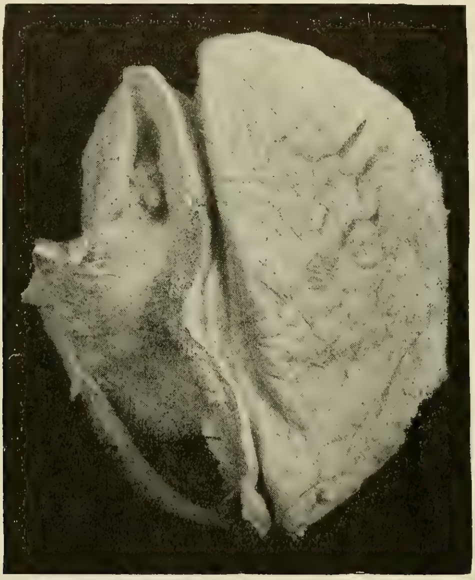
The acquired chronic form resembles so closely congenital internal hydrocephalus that we can consider the two together. So far as the name of the disease is concerned, the term hydrocephalus should be restricted to (1) congenital internal hydrocephalus and (2) chronic acquired internal hydrocephalus. In other words, there exists pathologically a certain class of effusions into the ventricles for which no cause is apparent. When these effusions reach a certain amount, the resulting symptoms are quite typical of what is called hydrocephalus, and clinically the term has therefore been confined to cases of this class.

PATHOLOGICAL ANATOMY.—The anatomical appearance of the brain itself, as a rule, corresponds with and may be accepted as the result of pressure by an increase in the intraventricular fluid. Fig. 282, page 844, represents the brain of a child who died of congenital internal hydrocephalus, and well exemplifies the pathology of the disease.

The convolutions are flattened and the walls of the ventricles are much thinned by the intraventricular pressure, while the ventricles themselves are much dilated. In some parts the cortex is less than 1 cm. ($\frac{3}{8}$ inch) in thickness. The amount of the fluid in these cases varies from a few cubic centimeters to three or four litres. The fluid has a specific gravity of about 1004.

The earlier the hydrocephalic condition begins, the larger the cranium will become. We therefore find the very large heads, as a rule, to be of the congenital variety. The head is at times of such a size as to cause difficulty in the delivery, or the fluid may collect very rapidly after birth, and the head soon assumes the characteristic appearance of hydrocephalus.

FIG. 282



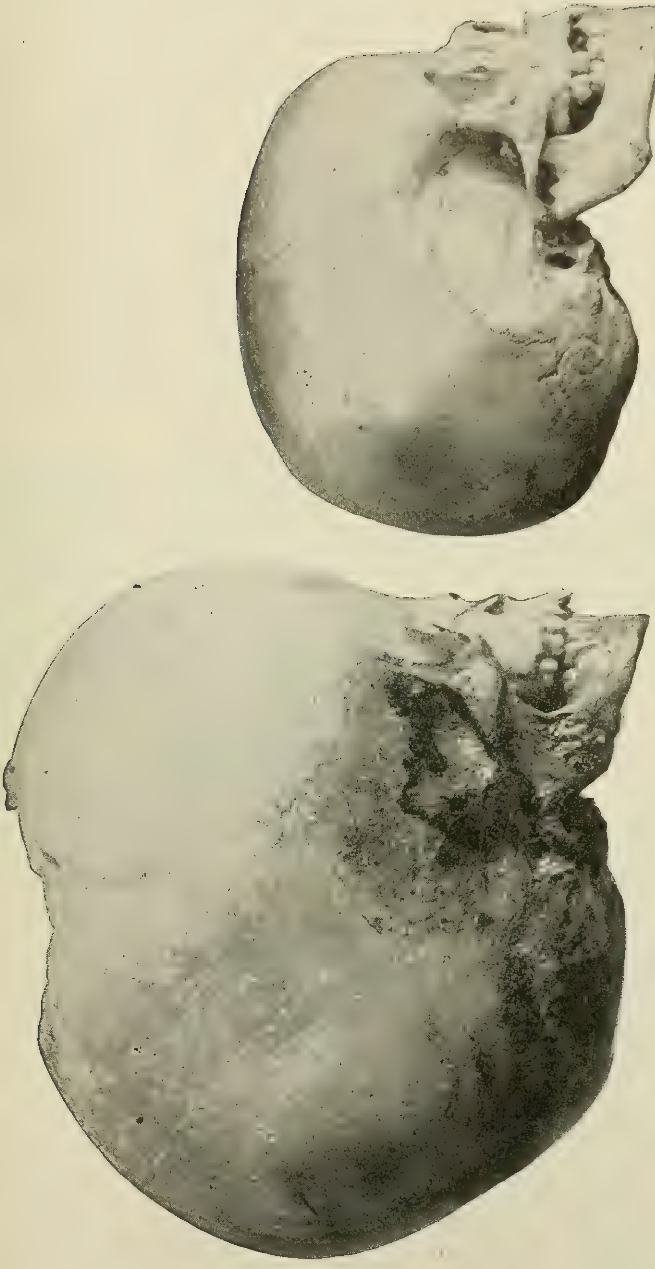
Hydrocephalic brain. Warren Museum. Harvard University.

Fig. 283 represents the hydrocephalic skull at three years in comparison with a normal skull of the same age.

The face in these cases of hydrocephalus remains about the same size as it would be normally, but usually looks much smaller from the disproportionate size of the cranium, which rests upon it like a globe.

SYMPTOMS.—The symptoms of congenital internal hydrocephalus are essentially those caused by pressure. We naturally, therefore, find the fontanels bulging and fluctuating, and the bones thin and forced out of position. The temporal and parietal bones diverge as they extend upward, while in the normal skull they ascend almost

FIG. 283



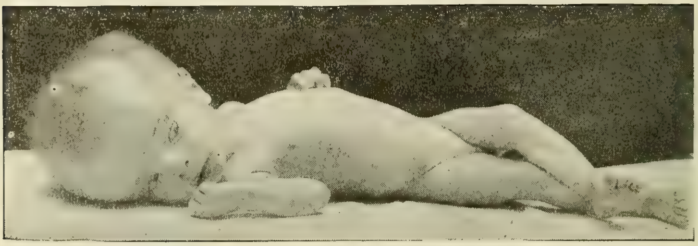
Hydrocephalic skull, child 3 years

Normal skull, child 3 years

Warren Museum, Harvard University

perpendicularly. This is shown in Fig. 284. If the disease has existed for some time, the upper wall of the orbit becomes flattened and the eyeballs protrude. The intracerebral pressure often produces a strong collateral circulation in the scalp and in the forehead, where the veins stand out like tortuous blue cords. Functional disturbances are numerous, and vary in almost every case. As a rule the children become idiotic, but at times, even in marked hydrocephalus, we find the mental condition normal, even when paralysis is present. As the various centers become affected by pressure, symptoms arise corresponding to the parts of the brain which are involved. Among these symptoms are nystagmus and, less frequently, strabismus. The pupils at first are usually moderately dilated. Later they become fixed, and sensibility to light is lost. The hearing lasts for a long time. The ability to walk is interfered with. Partial or general convulsions, paralysis (usually paraplegic), and contractures may occur. Pain in the head is often complained of, but, as a rule, is not so severe as in meningitis. There is difficulty in keeping the head erect, owing to its weight. The digestion is often good, and the appetite often voracious. The respiration is unaffected. The pulse is usually not retarded. The temperature, as a rule, is normal. The adipose tissue is sometimes abnormally increased. Even in mild cases, the eyes have a peculiarly characteristic down-looking expression.

FIG. 284



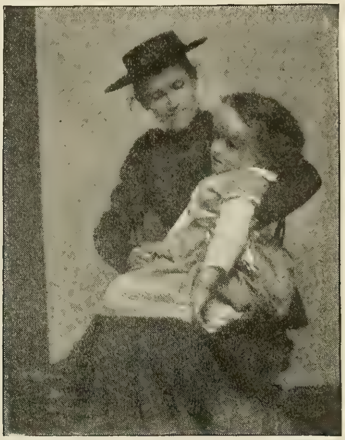
Congenital internal hydrocephalus. Male, 7 months old

DIAGNOSIS.—As congenital internal hydrocephalus is almost invariably attended by enlargement of the head and separation of the sutures, the diagnosis is not especially difficult, and is determined by comparing the measurements of the head with those of a normal head of the same age. It must be remembered, however, that hydrocephalus may exist with a small head, and that the general shape and circumference of the head in infancy vary in the individual to a considerable degree. When, however, these variations in size pass a certain limit or are combined with certain nervous phenomena they have a distinct pathological significance. In addition to the enlargement of the head, the symptoms of direct intracranial pressure make the diagnosis very simple.

PROGNOSIS.—The congenital cases, as a rule, die before childhood has been reached, but they have been known to live to middle age. Death usually occurs from some intercurrent affection. Complete recovery is very rare. Moderate hydrocephalus is compatible with good mental development.

TREATMENT.—The treatment of congenital internal hydrocephalus has been varied, but without marked success. As a certain number of cases are due to syphilis, antisyphilitic treatment should always be tried, as presenting the only hope. When the effusion is not large and is not increasing, moderate pressure with a rubber bandage seems to have a favorable result. When the disease is apparently not in an active state and is characterized by a very slight increase of fluid, aspiration through the anterior fontanel of a small quantity of fluid at a time has been of temporary or even permanent benefit. The point of aspiration should be 2 to 3 cm. ($\frac{5}{8}$ inch to $1\frac{1}{4}$ inches) from the median line, so as to avoid puncturing the longitudinal sinus.

FIG. 285



Congenital internal hydrocephalus.
Female, 5 years old

An operation for chronic hydrocephalus presents no technical difficulties. Of course only certain cases are suitable for operation. Moderate effusions should be let alone, also those cases in which a rudimentary development of the brain is suspected. Cases in which an operation is especially indicated are those which are both physically and mentally comparatively well developed up to the time when the enlargement of the cranium began. Such children should show the symptoms of direct intracranial pressure. They gradually become weak-minded or idiotic. They do not learn to talk, or they quickly forget what they have learned. They may also become totally blind. The power of walking is interfered with. Contractions and partial and general spasm are of ordinary occurrence. Unless the pressure is speedily removed, atrophy of the brain results, and if they live they remain idiots for life. It may be said that the results which have followed the operative treatment of hydrocephalus have not been encouraging.

Constriction of the scalp with adhesive plaster is useless. If there is great excitability bromide of potassium and chloral should be given in the form of enemata. Frequent lumbar puncture may also be resorted to, and it is generally to be preferred to the more radical practice of puncturing the ventricles. The fluid should be withdrawn guardedly, the pulse and respiration being watched with care.

PACHYMENINGITIS

This is an acute or chronic inflammation of the dura mater.

ACUTE PACHYMENINGITIS.—This is very rare in early life. It is most frequently seen in connection with middle ear disease. Some cases have followed trauma. The process begins as a localized inflammation of the outer layer of the dura. If the inflammation extends to the inner layer of the dura, the result is a leptomeningitis, which has been already described under Acute Meningitis. When the process remains confined to the outer layer of the dura, it may terminate in resolution, or in the formation of an abscess between the dura mater and the bone. The symptoms are entirely indistinguishable from those of cerebral abscess. The treatment is surgical.

CHRONIC PACHYMENINGITIS.—This condition is comparatively rare in early life. The cause is entirely unknown. Its chief clinical significance is that it is a frequent cause of meningeal hemorrhage. The symptoms are those which were described under hemorrhage of the brain. The diagnosis of the underlying chronic pachymeningitis cannot be made during life. The prognosis and treatment are essentially those of cerebral hemorrhage.

AMAUROTIC FAMILY IDIOCY

Amaurotic family idiocy is a comparatively rare disease characterized by blindness, mental deficiency, and spastic paralysis.

ETIOLOGY.—The disease is confined mainly if not wholly to the Jewish race. It shows a very marked familial occurrence, and often a number of children in the same family die from the disease. These are the only known facts concerning the etiology. The disease is apparently not congenital, as its time of development is not immediately after birth, but considerably later.

PATHOLOGICAL ANATOMY.—Both the brain and the spinal cord appear normal macroscopically, although the tissue seems somewhat more firm and elastic than normal tissue. On microscopic examination characteristic anatomical changes are found. The ganglion cells show a very marked degeneration, which is quite characteristic in appearance. There is swelling of the cells, lack of differentiation of the protoplasm and degeneration of the nucleus, which is excentrically situated. In the final stages the nerve cells disappear, and are replaced by neuroglia. The changes are very widespread, involving all parts of the central nervous system, including the retina.

SYMPTOMS. Infants with this disease usually develop normally until they are from six to ten months old. At about this time normal development, both physical and mental, appears to cease. At times it is noted that the eyesight is not as good as formerly, at other times the child simply appears to take less interest in its surroundings, and does not respond to stimuli. With the further progress of the disease, not only does normal development cease, but the child is obviously retrograding mentally and physically. There is progressive weakness of the muscles so that the power of sitting and holding up the head are lost, the eyesight becomes less and less good, and the child loses the power of recognizing faces and objects. He becomes more and more apathetic, and finally it is noticed that he is actually blind. In the later stages of the disease, the flaccid weakness of the muscles is replaced by rigidity, spasticity, and increased reflexes. Emaciation often does not become prominent until the later stages of the disease. Convulsions are seen at times.

On physical examination the most characteristic features of the disease are seen with the ophthalmoscope. In the place of the macula lutea there is a white or bluish-white area, in the center of which is a bright *cherry-red spot*. There is also atrophy of the optic discs. Both eyes are involved.

DIAGNOSIS.—The diagnosis depends upon the result of ophthalmoscopic examination. The cherry-red spot is diagnostic.

PROGNOSIS.—The outlook is extremely bad. The disease progresses steadily toward a fatal ending, and death usually occurs within a year of the first appearance of symptoms. At times, however, the baby may live in a blind, helpless and idiotic condition for several years.

TREATMENT.—No treatment is effective in this disease. The power of swallowing is often lost, and life can be prolonged only by resorting to tube feeding.

HEREDITARY ATAXIA

(Friedreich's Ataxia)

Hereditary or family ataxia is a comparatively rare disease, dependent on a slowly progressive sclerosis of the cord at different levels, which involves especially the posterior and lateral columns. It usually occurs in several members of a family, and develops in late childhood. The cause is unknown.

SYMPTOMS.—The characteristic symptoms are ataxia of the legs and arms, gradual loss of muscular power, with atrophy of the muscles, loss of the knee-jerks, disturbance of speech, and, in the late stages, nystagmus, muscular contractures from paralysis, giving rise especially to scoliosis and club-feet, and complete helplessness, with mental impairment.

A *cerebellar form* of the disease has been described by Marie, but this apparently does not develop until after the age of puberty.

DIAGNOSIS.—The disease is differentiated from tabes dorsalis by the absence of the crises and the Argyll-Robertson pupils of tabes, by the hereditary character of the affection, and the still greater rarity of tabes in early life.

It is differentiated from multiple sclerosis by the absence of increased reflexes, intention tremors, spastic gait, and ocular palsies.

PROGNOSIS AND TREATMENT.—The prognosis and treatment of hereditary ataxia is always unfavorable, and no treatment is of benefit except for the relief of symptoms.

JUVENILE TABES

(Locomotor Ataxia)

Cases of juvenile tabes are very rare. After a most painstaking search Mowburg of Vienna collected thirty-four cases, which, if critically analyzed may be reduced to twenty. The chief symptoms were loss of knee-jerks, Argyll-Robertson pupil, ataxia, lightning pains, bladder disturbance, primary progressive optic nerve atrophy, ptosis, girdle sensation and paresthesia. A study of these cases shows that in the juvenile form, vesical disturbance and optic nerve atrophy make their appearance early, while ataxia is either a late or absent symptom. Erb has maintained that juvenile tabes is always due to syphilis and later reported cases seem to support this view. The disease may show itself as early as the eighth or tenth year.

SYRINGOMYELIA

Syringomyelia is exceedingly rare in early life. It is regarded as a gliosis, a development of embryonal neuroglia tissue about the central canal of the spinal cord, in which hemorrhage and degeneration take place with the formation of cavities.

In this disease we usually find a diminution of sensation to heat and cold, involving the parts of the body below the site of the lesion, which is commonly a point in the upper dorsal or the lower cervical region. The sense of touch is preserved. There is apt to be a weakness of one or both arms, accompanied by marked wasting. There is usually some weakness in the legs. The reflexes are increased, and a spastic condition is likely to result. These symptoms are usually accompanied by marked lateral scoliosis.

Syringomyelia is an incurable disease, and the treatment is therefore usually limited to correcting, if possible, the lateral curvature which frequently accompanies it.

HEREDITARY SPASTIC PARALYSIS

The term hereditary spastic paralysis has been applied by Sachs to an hereditary disease characterized by spastic rigidity, chiefly of the lower extremities. He recognizes two types of the disease. One is the *spinal type*, a spastic paraplegia with contractures and increased reflexes, due to interference with the pyramidal tracts in the lateral columns of the cord. Another type, the *cerebral type*, develops at about four or five months of age. It is characterized by increasing mental dulness, defective vision ending in blindness, nystagmus, and gradual physical deterioration. Convulsions are never present. The symptoms may continue for one or two years, but the cases end fatally. The treatment is symptomatic.

FAMILY PERIODIC PARALYSIS

Family periodic paralysis is characterized by a periodic flaccid motor paralysis involving all the voluntary muscles except those of the face, eyes, tongue, speech, deglutition and the sphincters. The paralysis may be partial or complete and in certain seizures only the upper or lower extremities may be involved. Respiration may be difficult and the myocardium so weakened as to give rise to cardiac dilatation during the attack. There is complete absence of reflexes during a severe attack and the faradic irritability of the muscle is lost. The attacks may be precipitated by over-exercise. The onset is usually at night. Holtzapple has reported a family seventeen members of which were afflicted. In most of these cases the attacks began either at puberty or in the second decade. A few began as early as the eighth and ninth years.

During the periods between the seizures the patients are perfectly well. The attacks may last for hours or days, and the duration is uniform in a given case. The paralysis recedes in the reverse order of invasion.

PROGNOSIS AND TREATMENT.—Some cases outgrow the tendency to the attacks, while in the majority of cases the disease persists throughout life; it may end fatally from involvement of the muscles of the heart and respiration. There is no treatment which can be said to influence the course of the disease.

INSULAR OR DISSEMINATED SCLEROSIS

Insular or disseminated sclerosis is a chronic degenerative disease of the brain and cord, characterized by multiple distribution of areas of sclerosis.

ETIOLOGY.—The disease appears most frequently as a result of the specific infectious fevers, or of metallic poisoning. Heredity is of slight importance in the etiology, chiefly inasmuch as it predisposes to a neurotic constitution. The disease is most common in the second and third decades, but many cases in early life, even in infancy, have been reported.

PATHOLOGICAL ANATOMY.—The sclerotic patches may occur in the brain or cord or in both, and are very irregular in their distribution and size. The white matter of the brain, the pons, the medulla and the lateral columns in the dorsal and lumbar regions are most commonly involved. The sclerosed patches consist of proliferated neuroglia, the growth of which destroys the myelin of the nerves with but little destruction of the axis-cylinder.

SYMPTOMS.—The disease is generally slow in its development, and is characterized by tremors on attempting voluntary motions (*intention tremors*); by *scanning speech*, the words being pronounced slowly or with accentuation of the syllables and distinct tremulousness of the voice; by *nystagmus*, a rapid oscillatory movement of the eyes, especially when they are moved laterally, and by *spastic paralysis*, the gait being clumsy and staggering. The muscles are rigid and the deep reflexes increased. Ocular disturbances are common and are similar to those observed in hysteria. Inequality of the pupils and atrophy of the optic nerves may occur. In the majority of cases there is no disturbance of sensation.

As the disease progresses the speech becomes unintelligible, the memory fails, and the muscular weakness and paralysis is more and more marked until finally the patient is bedridden. Some cases are of more rapid development than others. Some may present bulbar symptoms in which not only the power of speech but of deglutition and respiration is affected. Apoplectiform or epileptiform seizures may occur in the course of the disease.

DIAGNOSIS.—A marked degree of muscular atrophy, and changes in the electrical reaction are not present in multiple sclerosis, and their absence is of much aid in the differential diagnosis.

Multiple sclerosis may be distinguished from transverse myelitis by its very gradual onset and the absence of sensory symptoms.

The diagnosis from hereditary ataxia was given under that disease. The nystagmus, scanning speech, and intention tremor will serve to distinguish the disease from hysteria, which it may at times resemble. Chorea may be eliminated in the diagnosis by the absence of the characteristic incöordinate movements, and by the presence of nystagmus and true ataxia.

PROGNOSIS.—The disease is incurable, and may last for years. Remission of the symptoms and even improvement may occur, but the general course of the disease is progressive.

TREATMENT.—Rest in bed, hydrotherapeutics, massage, and general hygiene are desirable in that they alleviate the condition, rendering life more bearable, but no treatment can be said to be in any way curative.

MULTIPLE NEURITIS

Neuritis is an inflammation of the peripheral nerves. It is accompanied by pain and tenderness in the affected regions, and in the more severe cases by paralysis and atrophy. Neuritis of a single nerve-trunk or of its branches may be caused by traumatism, cold, or pressure, or may occur as a manifestation of toxemia in the course of various diseases. In certain constitutional conditions a number of nerves in different parts of the body are affected, constituting the disease called *multiple neuritis.*

ETIOLOGY.—Multiple neuritis in early life occurs usually in the course of or subsequent to one of the infectious diseases. Of these diphtheria is the most common, but in rare cases it is seen to follow scarlet fever and measles. A mild form sometimes occurs after typhoid fever. At times multiple neuritis is produced by drugs, such as lead, arsenic, or alcohol. This form is not common among children. The epidemic form of the disease, known by the terms kakke and beri-beri, is quite rare in this country.

PATHOLOGICAL ANATOMY.—The pathological condition in multiple neuritis is an interstitial inflammation or parenchymatous degeneration. A few nerves may be affected, or the distribution may be general. The cranial nerves are rarely affected.

SYMPTOMS.—The onset of the disease may be acute or subacute, but in most cases is gradual. It may at the beginning present severe symptoms, such as extreme pain, tenderness over the nerve-trunks, and fever with an accompanying paralysis. On the other hand, the pain in the beginning may be very slight, and the first symptoms noted may be a gradually increasing weakness of the limbs, while the tenderness may be found only when especially sought for. There may be hyperesthesia, anesthesia, numbness, and loss of muscular power. Both the sensory and the motor nerves are affected and the condition is symmetrical. After the acute symptoms have passed away the faradic irritability is diminished; the reaction of the nerves to the galvanic current is diminished, and the reaction of degeneration develops. When the extensors of the leg are involved there is foot-drop, and when those of the forearm are affected there is wrist-drop. Rarely all the muscles of the extremities may be paralyzed, and the child becomes perfectly helpless. The course of the disease is apt to be a long one of several months, and in the later stages atrophy occurs, while the early hyperesthesia may give place to a more or less marked anesthesia, and numbness and various other paresthesiae may occur. In the mild cases, where only pain and tenderness exist, the knee-jerks may not be diminished, and in the early stages may even be slightly increased, but in more typical cases of the disease they are absent. Early contractures and spasmodic conditions are absent, the paralysis being flaccid. The bladder and rectum are not affected. The temperature is apt to be somewhat elevated, and is decidedly so at the onset when the disease is acute. The sensory symptoms are most marked in the beginning of the attack and subside more rapidly than the motor.

DIAGNOSIS.—The diagnosis depends on the fact that symmetrical nerve areas are affected by both sensory and motor symptoms, and that there is tenderness of the nerve trunks. The pain and tenderness associated with anterior poliomyelitis may be due to some degree of neuritis, but these cases do not present the anesthesia and paresthesia of the advanced form of neuritis. Moreover, the paralyses in multiple neuritis are symmetrical. Both diseases generally show the reaction of degeneration.

The disease might in some cases be mistaken for the form of acute ascending paralysis known as *Landry's paralysis*. This is, however, a form of paralysis due to a poliomyeloencephalitis, which begins in the lower extremities and involves successively the abdominal, thoracic and arm muscles, usually terminating in death from paralysis of respiration.

PROGNOSIS.—The prognosis of multiple neuritis is favorable even when the disease begins with an acute onset accompanied by delirium and high fever, and, although the paralysis may last for many months, the cases usually recover. Exceptionally, however, the results may be permanent, and death may take place from paralysis of the heart or of the muscles of deglutition and respiration. When the reaction of degeneration is present the prognosis for complete recovery is not so good and the case is apt to be prolonged.

TREATMENT.—The treatment is at first absolute rest in bed, and later electricity, massage, and strychnin. The primary cause of the disease having been determined, it should, if possible, be removed. In cases of obscure etiology, the urine should be examined for arsenic or lead, and the case treated accordingly.

The treatment of those cases which follow the acute infectious diseases is symptomatic and hygienic. For the pain the application of heat is the most efficacious remedy. In the acute cases electricity should not be begun until after three or four weeks, or until acute symptoms have subsided; faradism should be used if a reaction is obtained with a moderate current, otherwise galvanism. Iodid of potassium is indicated in those cases which are caused by lead or arsenic. In the subacute cases electricity and massage should be employed from the very beginning. Strychnin is usually the only medication used, but I do not think it has much influence on the disease.

PROGRESSIVE MUSCULAR ATROPHY

Progressive muscular atrophy of central origin, also described as the *Aran-Duchenne* type, develops in the great majority of cases in adult life after the twenty-fifth year. A few cases, however, have been reported in young children, notably two by Hoffman, one of which occurred in a girl four years of age, and another in her brother, in whom the symptoms began at about the same age.

Various forms are recognized in adults and are known as *amyotrophies*, by which is meant a progressive wasting due to lesions in the spinal cord, as distinguished from the term *myopathies* or *dystrophies*, in which the primary disease is in the muscles themselves. Midway between these two types of muscular atrophies, both in the pathological lesions and clinical symptoms, is another, which is described as *progressive neural muscular atrophy*.

ETIOLOGY.—The direct cause of the progressive muscular atrophy of central origin is not known. Hereditary and family influences are, in adults, of much less importance in relation to this spinal type of progressive muscular atrophies than they are in the muscular atrophies of neural origin and in the dystrophies. The association of the disease, however, in the children of one family has been noted, and is a characteristic of the infantile form. Trauma, exposure to wet, cold, fright, and nervous shock are mentioned as possible etiological factors.

PATHOLOGICAL ANATOMY.—The pathological lesions consist primarily in a slow degeneration of the lower neurons of the motor path, resulting in progressive atrophy of certain groups of muscles. The chief histological changes consist of an atrophy of the ganglion-cells of the anterior horns and an increase in the neuroglia. The lateral pyramidal tracts may show degenerative changes, which, in some cases, have been traced to the motor cortex, and even to the cortical motor cells themselves. When this is marked the disease is called amyotrophic lateral sclerosis. The direct cerebellar and ventrolateral tracts are not involved. The muscles and the intermuscular branches of the motor nerves show degenerative changes.

SYMPTOMS.—Indefinite pains suggesting those of chronic rheumatism may be the earliest symptoms. These are followed by a gradual loss of power and atrophy of the muscles. The muscles of the thumb, both in the thenar and hypothenar groups, and then the interossei and lumbricales are first affected, leaving depressions between the metacarpal bones. The wasting process then attacks the flexor and extensor muscles of the forearm, contractures set in, and the characteristic " claw-hand " develops. The deltoid is the first of the shoulder muscles to atrophy, and then follow in the typical cases the remaining muscles of the upper extremity, the muscles of the trunk, and finally those of the lower extremity. This order of progression is not always maintained. In rare cases the legs may begin to atrophy soon after the affection of the hand. Muscular contractures from the action of antagonistic muscles, fibrillary twitching in the unaffected muscles, numbness and coldness in the regions of atrophy, and diminution and even loss of muscular reaction to faradic and galvanic currents are generally present in advanced cases. The reaction of degeneration may be present in the muscles in which the atrophy is extreme. The excitability of the nerve-trunks outlasts that of the muscles. The muscular weakness is proportionate to and generally dependent upon the degree of atrophy. The sensations of heat, touch, and pain are not impaired.

In another type of cases, described by Charcot as *amyotrophic lateral sclerosis*, a spastic paralysis is associated with the wasting and is due to degeneration of the pyramidal tracts, but so far as we know this condition does not occur in children.

In very rare cases in children the degenerative processes may affect the nuclei of the motor cranial nerves, both the upper and lower divisions of the motor tract being involved (Sachs). The symptoms in these cases bear a close resemblance to the progressive bulbar paralyses of adults, but are so rare that they need merely be referred to.

DIAGNOSIS.—The diagnosis is made from the slow, progressive wasting of muscles in the order described, the gradual loss of power in the affected groups, the fibrillary muscular contractions, the diminution in the electrical reactions, the absence of marked impairment of sensation, and the absence of hereditary or family influences. The rarity of the disease in early life is also of some value in the diagnosis. It is to be distinguished from ulnar neuritis and syringomyelia by the absence of disturbance of sensation.

PROGNOSIS.—The disease is progressive and may last for many years. It is invariably fatal, although there may be periods in which there is an apparent arrest of the process, but this is only temporary.

TREATMENT.—The treatment is at best palliative and for the most part ineffective. Massage, electricity, anti-specific treatment if there is any evidence of a syphilitic taint, arsenic, and hypodermic injections of strichnin, as recommended by Powers, may be tried.

End of Volume III

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